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ALBAMYCIN* IN THE TREATMENT OF SURGICAL INFECTIONS

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The following report is concerned with the use of Novobiocin or Albamycin (Upjohn) in the treatment of 36 patients with infection and 4 patients with potential infection encountered in the general surgical service of the Medical Center Hospitals.

INTRODUCTION

Albamycin is a relatively new antibiotic produced by *Streptomyces niveus*, first isolated from soil. Since its introduction, Albamycin has been found to be particularly effective in the treatment of Gram positive cocci infections, especially those due to staphylococci, as well as infections due to certain Gram negative organisms, notably proteus.[1-3, 4, 6] Of special interest is the reported efficacy of the drug against staphylococcal organisms resistant to other antibiotics.[5]

Albamycin may be administered either orally, intramuscularly, or intravenously. The usual dose ranges between 250 to 500 mg. every 6 hr. whereas the recommended dosage for parenteral use in adults is 500 mg. every 12 hr.; 15 mg. per kg. of body weight is the usual parenteral dose given to children. Peak serum levels of the drug occur 2 to 4 hr. after oral ingestion, and unusually high and prolonged serum levels have been detected for 24 hr. and longer.[7] Sustained serum levels are probably due in part to reabsorption of the drug (since approximately one-third is excreted in the bile) as well as to loose binding of the drug by serum proteins. Experimental studies in animals have demonstrated that the drug diffuses readily into pleural and ascitic fluid, but is not found in normal cerebrospinal fluid. Only small amounts are excreted in the urine; however, the concentration appears to be sufficient to inhibit growth of organisms found *in vitro*.

Side reactions to Albamycin are reportedly few in number, the most common being the development of urticaria or a maculopapular dermatitis. The incidence of skin reactions in adults receiving 1.0 gm. per day for a total of 10 gm. has been reported as 0.5 per cent, although the incidence increases with higher dosage.[2] Additional side effects that have been reported include gastrointestinal disturbances (nausea, vomiting, diarrhea), leukopenia, neutropenia, and eosinophilia. All side effects are said to disappear rapidly after discontinuing the drug. A yellow pigment, thought to represent a metabolic by-product of the drug, has been detected in the plasma of some patients during treatment. The presence of this pigment may interfere with serum bilirubin and icterus index determinations, although it has not been shown to be associated with liver damage.

CLINICAL STUDY

Methods. Albamycin was used in the treatment of 36 patients with infection encountered in the general surgical service. In addition, the drug was used prophylactically in 4 patients during the postoperative period. Albamycin was administered orally in 31 patients, intramuscularly in 1 patient, and both orally and intravenously in 8 patients. In the majority of instances, the oral dose was 250 mg. every 6 hr. and the parenteral dose 500 mg. every 12 hr. Cultures were obtained in 28 of the 36 patients with infection, and

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in vitro sensitivity tests were performed in all but 2 of these patients. Sensitivity of organisms to antibiotics was determined by the amount of colony growth occurring around commercially prepared antibiotic discs. The organisms were graded as very sensitive, moderately sensitive, slightly sensitive, or resistant to the antibiotic being tested.

Results. In 26 patients there was marked improvement which was thought to be definitely related to the use of Albamycin. Improvement in 4 additional patients was believed to be due primarily to local treatment (incision and drainage, warm saline soaks, mechanical debridement, etc.), rather than to the effects of the antibiotic. Six patients in this series failed to improve after Albamycin therapy. Two of these patients had advanced arteriosclerosis obliterans and failure to respond to therapy was undoubtedly due in part to deficient blood supply to the infected area. Moreover, lack of improvement in 1 of these patients, as well as in 2 others, may have been the result of insensitivity of the invading organisms to the antibiotic because the organisms isolated by culture from these patients were shown to be resistant to Novobiocin *in vitro*. The failure to improve in 2 additional patients was thought to be the result of inadequate drainage from the infected areas. This factor was demonstrable in 1 of these patients, with a lung abscess, who improved rapidly after adequate drainage had been established by rib resection and incision of the abscess cavity. In the other patient, with carcinoma of the lung and associated pneumonitis, the persistent bronchial obstruction is thought to have precluded any response to antibiotic therapy. In all 4 patients in whom Albamycin was used prophylactically in the postoperative period, no evidence of infection occurred.

TABLE I

In vitro sensitivity of various isolated strains of bacteria to Novobiocin

Organism	Strains Tested	Very Sensitive	Moderately Sensitive	Slightly Sensitive	Resistant
	no.				
1. *Staphylococcus aureus*	8	7			1
2. *Staphylococcus albus*	3	3			
3. *Micrococcus* sp.	2	1	1		
4. α-hemolytic streptococcus	15	14			1
5. β-hemolytic streptococcus	2	1			1
6. Nonhemolytic streptococcus	4	2			2
7. *Pneumococcus* sp.	2	2			
8. *Proteus* sp.	7	1			6
9. *Aerobacter Aerogenes*	3				3
10. *Alcaligenes* sp.	4	3			1
11. *Bacillus subtilis*	1	1			
12. *Escherichia coli*	3	1			2
13. *Paracolon* sp.	1	1			
14. *Pseudomonas* sp.	3				3
15. *Salmonella paratyphi* "B"	1				1
16. Neisseria	7	6			1
17. Diphtheroids	3	3			

Side effects were observed in 6 patients during the course of treatment. Two patients developed a skin rash, one urticarial in type and the other morbilliform, after total oral doses of 9.0 gm. and 4.25 gm., respectively. Pruritus was present in both patients. Eosinophilia (greater than 10 per cent) was detected in 1 of the patients with a skin rash, total dose 4.25 gm., and in 2 other patients after receiving total doses of 19.25 gm. orally and 1.0 gm. intravenously. A mild diarrhea developed in 1 patient, in whom the drug was used prophylactically, after receiving 3.75 gm. orally. Leukopenia or neutropenia was not detected in any patient during the course of treatment. Pruritus associated with the skin rashes was well controlled with antihistamines, and all side effects disappeared over a period of two to three days after discontinuing the antibiotic.

DISCUSSION

Although no definite conclusions can be drawn from this series of cases, certain impressions are gained regarding the effectiveness and side effects of Albamycin. A review of *in vitro* sensitivity tests performed on organisms isolated from patients in this study (table I) shows that the drug is effective *in vitro* against Gram positive cocci with no significant influence on proteus organisms. In this series, six out of seven strains of Proteus proved resistant to the drug. Clinically these results were substantiated by improvement in a significant number of patients with Gram positive cocci infections. In view of this series and others previously reported,[1-3, 4, 6] it would seem preferable to reserve the use of Albamycin

for Gram positive cocci infections, in particular those due to staphylococcal organisms resistant to other antibiotics. Side effects of Albamycin, which seem to occur in a relatively small percentage of patients (1.5 per cent in this series), are easy to control, and disappear within two to three days after discontinuing therapy.

SUMMARY

1. A brief review of the pharmacologic properties of Novobiocin or Albamycin (Upjohn) is presented.

2. Thirty-six cases of infections encountered in the general surgical service of the Medical Center Hospitals were treated with oral, intramuscular or intravenous Albamycin. Improvement thought to be definitely related to the use of Albamycin was noted in 26 cases.

3. In 4 cases, Albamycin was used prophylactically in the postoperative period, and no evidence of infection occurred.

4. Side effects which developed in a small percentage of cases (1.5 per cent) were easy to control and disappeared within two to three days after discontinuing antibiotic therapy.

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SUBCUTANEOUS SPACE CONTAMINATION

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INTRODUCTION

Infections in so-called "clean" surgical wounds are a tremendous problem for surgeons and patients in whom they develop. When he has followed strict surgical mandates concerning sterility, the surgeon is at a loss to explain these occurrences. Moreover, the patient is unable to comprehend the development of a wound infection in an area which was never contaminated or "dirty" before the operation. Many times, the surgeon has to decide whether to use antibiotics postoperatively in clean wounds or to wait for developments.[4] This study was not undertaken in an effort to answer the latter problem but simply to make some observations on the results of bacterial cultures of the subcutaneous tissue at the operating room table, and to assess the value of irrigation with isotonic saline solution and neomycin sulfate solution.

MATERIALS AND METHODS

The patients selected for this study were 102 private surgical cases of the author. The prerequisite was that there should be no preoperative infection of the tissues to be attacked. If a patient turned out to be contaminated by any inflictions such as peritonitis, abscess, etc., he was not included in this study. Otherwise there was no selection of patients from the standpoint of age, sex, type of operation, type of incision, region of incision, time of day, anesthesia, suture material, or extraneous sources of contamination (operating room, assistants, nurses, etc.). All bacterial cultures were taken by the author under sterile precautions at the operating room table. The subcutaneous space was the only source of the culture material. Table I lists the types of operations performed in this series. All wounds were carefully draped with towels which were placed in such a way as to completely exclude the skin around the entire wound. If the towels became moist they were changed. Swabs were taken and immediately sent to the bacteriology laboratory to avoid drying or contamination. In the bacteriology laboratory the cultures were plated for both aerobic and anaerobic growth. Sensitivity studies were carried out for penicillin, streptomycin, Terramycin, tetracycline, Aureomycin, erythromycin, Chloromycetin, triple sulfa, sulfadiazine and neomycin.

Saline solution used for irrigation in these cases was obtained from the scrub-up nurse's instrument table. There was no attempt to use a separate saline solution. It was thought wise to use the ordinary facilities of the operating room table.

The wound was irrigated with 0.5 per cent neomycin sulfate solution (0.5 gm. diluted in 100 cc. normal saline), using an ordinary Dakin's syringe.

Cultures of the subcutaneous space were taken between 15 and 45 min. after the initial incision had been made. Some wounds were irrigated with normal saline solution, after which repeat cultures were taken and other wounds were irrigated with neomycin sulfate solution, following which cultures were taken. Some wounds were irrigated with both normal saline solutions and neomycin sulfate solution.

FINDINGS

Of the 102 subcutaneous spaces cultured prior to any irrigation, 43 (42.2 per cent) revealed no bacterial growth, and 59 (57.8 per cent) yielded positive cultures (table II). The most common organism found was the hemolytic *Staphylococcus aureus*. The hemolytic *Staphylococcus albus* and the *bacillus* sp. and the nonhemolytic *S. albus* were also frequently found.

In general, the organisms were sensitive to many of the drugs studied. However, every organism cultured was sensitive to Chloromycetin and neomycin. There were only three instances in which the organisms were not sensitive to erythromycin and two additional instances in which the organisms were only weakly sensitive to erythromycin.

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TABLE I

Types of operations

	no.
Hernia repair (Incisional, inguinal, umbilical, diaphragmatic)	28
Breast (Excision of tumor, simple mastectomy, radical mastectomy)	19
Benign tumors and cysts (excisions)	18
Thyroidectomy	13
Phlebectomy (saphenous)	8
Cholecystectomy	3
Laparotomy	3
Thyroglossal cyst	2
Gastric resection	2
Amputation	1
Aneurysmorrhaphy	1
Tenorrhaphy	1
Parotidectomy	1
Lymphadenectomy	1
Splenectomy	1
Total	102

TABLE II

Subcutaneous space cultures (102 cases)

	Direct Subcutaneous	After Saline Irrigation	After Neomycin Irrigation
Number of times subcutaneous space was cultured	102	55	81
Number of times positive for:			
Hemolytic *Staphylococcus aureus*	25	17	2
Hemolytic *Staphylococcus albus*	17	10	1
Bacillus sp.	12	5	2
Bacillus subtilis	4	2	2
Sarcina	2	3	3
Micrococcus sp.	3	6	1
Nonhemolytic *Staphylococcus aureus*	2		1
Nonhemolytic *Staphylococcus albus*	7	2	1
Anaerobic micrococci	1		1
Neisseria		1	
Corynebacterium hoffmannii	5	1	
Diphtheroids	1	2	4
Aerobacter Aerogenes	2		
α-streptococci		3	
γ-streptococci		1	
Streptococcus fecalis	1		
Yeast			1
Clostridium welchii		1	
Pseudomonas aeruginosa	1		
Proteus			1
Flavobacterium bacillus	1		
No growth	43	20	65

Is the subcutaneous space contaminated? The subcutaneous space which was cultured directly on 102 occasions was bacteriologically uncontaminated in 43 instances (42.2 per cent). The subcutaneous space was bacteriologically contaminated in 57.8 per cent of the patients. In view of the fact that the cultures were taken from the wounds very shortly after the initial incision and draping were completed, one may state that the length of the operative procedure lends no aid in predicting which subcutaneous space will be contaminated.

Role of saline irrigation. Saline irrigation was used for 55 patients. In 22 of these (40 per cent) the wounds were recorded as bacteriologically contaminated, as determined by the original direct subcutaneous cultures. They remained so after the saline irrigations. In 14 patients (25.4 per cent) the subcutaneous space was uncontaminated originally and remained so after saline irrigation. In 8 patients (14.5 per cent) the direct subcutaneous space was positive on culture before saline but revealed no growth after saline irrigation. In 10 patients (18.2 per cent) the subcutaneous space was bacteriologically uncontaminated before saline irrigations but revealed positive bacteriologic cultures following saline irrigations. In view of this small series it is not possible to state categorically that the saline solution itself might be contaminated. This possibility does arise, however, especially in consideration of other studies which have been published which show the occurrence of positive cultures in alcohol basins, saline basins and soap dishes.

Role of neomycin irrigation. In this series of 102 cases the subcutaneous space was irrigated with neomycin sulfate solution on 81 separate occasions. Approximately 100 cc. of a 0.5 per cent solution was used to irrigate the wounds before culturing them. The maximum used on any one wound was 0.5 gm. In 29 patients (35.8 per cent) the subcutaneous space was bacteriologically uncontaminated before and after irrigation with

neomycin solution. In 32 patients (39.5 per cent) the subcutaneous tissue was bacteriologically contaminated prior to the neomycin irrigation but negative to culture after the neomycin irrigation. In 12 patients (14.8 per cent) the subcutaneous space was bacteriologically contaminated before irrigation with neomycin and remained so after irrigation. In 6 patients (7.4 per cent) the subcutaneous tissues were uncontaminated prior to neomycin irrigation and bacteriologically positive after its use. None of the 81 wounds irrigated with neomycin sulfate solution showed any indication of ototoxicity, nephrotoxicity or respiratory arrest.[1-3, 5, 6]

SUBSEQUENT WOUND INFECTIONS

None of the cases studied revealed evidence of a wound infection. All of these cases were carried on the appropriate antibiotic as determined by the cultures and drug sensitivity studies. Therefore, from this study it is difficult to make any statement regarding the potentialities of a bacteriologically contaminated subcutaneous space in terms of anticipated wound infection. Other observations of the author showed that the organisms most frequently cultured in wound infections developing in clean operative wounds were hemolytic *S. aureus* and *S. albus*. In view of this, it would seem desirable to use antibiotics prophylactically in cases where there is a contaminated subcutaneous space.

SUMMARY

1. The results of bacterial cultures from the subcutaneous tissue of 102 unselected clean surgical wounds are presented.

2. The factor of positive bacterial cultures, after irrigating these wounds with either normal saline solution or 0.5 per cent neomycin sulfate solution, is demonstrated.

3. Bacteriologically contaminated wounds (57.8 per cent of the cases) became sterile in 27 per cent (8 out of 30) of the cases following saline solution irrigations, and in 73 per cent (32 out of 44) of the cases following irrigation with neomycin solution.

4. All of the organisms cultured were sensitive to Chloromycetin and neomycin.

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OBSTRUCTION OF THE DISTAL DUODENUM DUE TO INTRAMURAL HEMORRHAGE*

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Mechanical obstruction of the second and third portions of the duodenum is not encountered frequently. Many disorders can produce obstruction of this portion of the duodenum, the most frequent being congenital anomalies, arteriomesenteric occlusion, peptic ulceration, impaction by gallstones and foreign bodies, benign and malignant tumors of the duodenum, and neoplasms of adjacent structures. Obstruction of this portion of the gastrointestinal tract by intramural hemorrhage or hematoma in the duodenal wall is rare. In reviewing the medical literature, reports of 9 cases have been found. Within a year the authors have observed 2 patients who developed an obstruction of the duodenum distal to the ampulla of Vater, resulting from an intramural hemorrhage.

CASE REPORTS

DCGH #E 49523: A four-year-old colored female was admitted to the District of Columbia General Hospital on February 8, 1955, with the chief complaint of anorexia, nausea, and vomiting of one day's duration. Three days before admission, the child fell backwards off a chair, and suffered a fracture of the left radius and ulna. She was seen in the emergency room of the hospital, where the fracture was reduced and a cast applied. A general physical examination did not reveal other injuries. She was sent home. She remained well for two days until she lost her appetite and began to vomit. The remainder of the history was noncontributory.

Physical Examination. T. 99.4°F.; P. 160; B. P. 64/45 mm. Hg. The patient was a well developed, pale, somewhat dehydrated colored female appearing acutely ill. The abdomen showed no distention; there was a slight feeling of fullness on the left side of the abdomen but no definite mass. There was no spasm or rebound tenderness present. Peristalsis was absent. The remainder of the examination was negative.

The admission blood count showed leukocytes 2,800 per cubic mm. with 73 segmented cells and 7 band cells. The hematocrit was 30. Roentgen examination of the abdomen showed gaseous distention of the stomach and first portion of the duodenum with no gas distal to this point.

Treatment was begun with gastric decompression by nasogastric suction. Anemia and dehydration were corrected. After 48 hr. nasogastric suction was discontinued, but in a few hours the child began to vomit again. On the third hospital day an upper gastrointestinal series was made which demonstrated an obstruction of the barium flow through the duodenum (fig. 1).

On the fourth hospital day the patient was explored through a right upper paramedian incision. There was old blood present in the peritoneal cavity. Exploration revealed a retroperitoneal hematoma involving the head of the pancreas and extending into the walls of the second and third portions of the duodenum. The serosa of the duodenum was incised and the hematoma was evacuated. A drain was inserted in the area and the abdomen was closed. The postoperative course was uneventful. The child had no difficulty when oral feeding was begun. Follow-up examinations have shown no further evidence of obstruction.

GWUH #97572. The patient was a 38-year-old colored female, domestic, admitted to the George Washington University Hospital medical service on February 1, 1956, with the chief complaint of nausea and vomiting of three days' duration. The patient had been in good health until three days prior to admission, when she experienced the sudden onset of sharp colicky periumbilical pain and vomiting. On the second day of illness the pain gradually subsided, but nausea and vomiting continued. She came to the hospital the third day of illness because of persistent vomiting. The patient gave a history of heavy drinking prior to the onset of illness. She had not experienced similar attacks before.

Physical Examination. Examination revealed the patient to be a well developed, well nourished, colored female, moderately dehydrated, alert, cooperative, and not in acute distress. T. 100°F.; P. 120; B. P. 120/85 mm. Hg. The abdomen was slightly distended, but not tense. There was no

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FIG. 1

guarding or rebound tenderness. There was slight tenderness on palpation to the right of the umbilicus. The remainder of the physical examination was not remarkable.

The admission blood count showed leukocytes 13,400 per cubic mm.; segmented cells 79; band cells 2. Serum amylase and lipase were at the upper limits of normal. Blood urea nitrogen was 41 mg. per cent.

After admission the patient continued to vomit. Nasogastric suction was instituted, and 2,000 cc. of foul-smelling, yellow-green guaiac-negative material was aspirated from the stomach. Gastrointestinal decompression was continued and parenteral fluids were administered. On the seventh hospital day a gastrointestinal series was made and repeated on the ninth day. Both examinations showed the second portion of the duodenum to be displaced by what appeared to be an extrinsic mass (fig. 2, *A* and *B*).

The patient was transferred to the surgical service and explored through a right upper paramedian incision. Upon entering the peritoneum, approximately 500 cc. of old blood was found free in the peritoneal cavity. There was some infiltration of blood in the wall of the ascending colon and the transverse mesocolon. Further exploration showed the head of the pancreas to be ecchymotic and indurated. Fat necrosis was not encountered, but because of the swollen indurated pancreas it was believed that the patient was suffering from acute pancreatitis. The duodenum was inspected. The second and third portions were enlarged to more than twice their normal diameter and the wall was surrounded by a subserosal infiltration of blood. The second portion was rotated medially and palpated. The lumen was almost completely occluded by intramural blood. No specific procedure was done. The abdomen was closed.

Postoperatively the patient did well. The gastric tube was removed on the third postoperative day, when she tolerated a graduated diet.

DISCUSSION

Table I summarizes the pertinent data of the 9 cases previously reported in the literature in addition to 1 case contributed by Dr. Schreiber[4]

FIG. 2: *A*, left; *B*, right

TABLE I

Author	Age, Sex	Etiology	X-Ray Finding	Operative Finding	Operative Procedure
Oppenheimer[5]	42 yr., female	Pancreatitis and carcinoma	None taken	Submucosal hematoma, 2nd and 3rd part	Drainage
Schreiber[6]	6 yr., male	Abdominal trauma	None taken	Hematoma, 2nd part	Evacuated
Dey[1]	6 yr., male	None apparent	None taken	60-cc. hematoma, 2nd part	Evacuated
Stirk[7]	9 yr., male	Abdominal trauma		114-cc. clot, 2nd, 3rd, 4th part	Evacuated
Magladry and Mathewson[3]	9 yr., male	Abdominal trauma	Obstruction, 2nd part	Hematoma, 2nd part	Evacuated
Felson and Levin[2]	18 yr., male	Abdominal trauma	Intramural extramucosal defect	20-cm. hematoma, 2nd and 3rd part	Evacuated
Felson and Levin	9 yr., female	Abdominal trauma	Coil spring pattern	Not explored	None
Felson and Levin	33 yr., male	None apparent	Coil spring pattern	7-cm. clot, 3rd part	Evacuated
Felson and Levin	9 yr., male	Abdominal trauma	Coil spring pattern	45-cc. clot, 3rd part	Evacuated
Melamed and Pantone[4]	19 yr., female	Abdominal trauma	Suggested intramural tumor	Not explored	None
McClelland *et al.*	4 yr., female	Abdominal trauma	Coil spring pattern	Hematoma, 2nd and 3rd part	Evacuated
McClelland *et al.*	38 yr., female	Pancreatitis	Coil spring pattern	Hematoma, 2nd and 3rd part	Exploration only

of Sidney, Australia, and the 2 cases presented in this paper.

This condition seems to occur principally in children and young adults. Trauma was the etiology in 8 of the 12 patients. A relatively mild degree of abdominal trauma was responsible for this condition in almost every instance since most of the patients in this group were able to continue their normal activities after injury. Symptoms of obstruction did not develop and hospitalization was not required until hours or days after the trauma occurred.

Pancreatitis and carcinoma was the etiology in the patient presented by Oppenheimer.[5] In the authors' second patient, the etiology was thought to be pancreatitis alone. Although the serum amylase level was at the upper limits of normal on the second hospital day, this was four days after the onset of illness. This, along with the history of alcoholism and the findings at laparotomy, led us to believe that it is the only patient in the series in which pancreatitis was the sole etiology. In 2 patients the etiology was unknown.

In 1954 Felson and Levin[2] described an x-ray configuration in 3 of their 4 patients which they believed to be characteristic of intramural hemorrhage in the second and third parts of the duodenum. They described a coil spring pattern in the duodenum with barium study. They believed it to be caused by crowding and stretching of the valvulae conniventes from the blood in the duodenal wall. It is of interest that the 2 patients presented in this paper demonstrated the coil spring pattern described by Felson and Levin.

Of the 12 patients recorded, 2 were not surgically explored, the diagnosis having been made by finding the typical coil spring pattern or intramural defect of the duodenum on x-ray examination. One patient was subjected to laparotomy only. In 9 patients the intramural and retroperitoneal hematomas were evacuated or drained.

There was 1 death in the group. This was Oppenheimer's patient, who died 30 min. after operation, apparently of shock. In all the other patients there was an uneventful postoperative course with no further evidence of obstruction.

SUMMARY AND CONCLUSION

1. Two cases of obstruction of the second and third portion of the duodenum due to intramural hemorrhage are presented.

2. Ten previously reported cases are summarized.

3. The coil spring pattern demonstrated by upper gastrointestinal roentgenograms described previously by Felson and Levin is confirmed in the author's 2 cases.

4. In most instances proper management consists of operative intervention and evacuation of the subserosal collections of blood in the duodenum.

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CONGENITAL DEFICIENCY OF ABDOMINAL MUSCULATURE (A SYNDROME): A SURGICAL REPAIR*

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Congenital deficiency of the abdominal musculature is uncommon. Less than 70 cases have appeared[10, 23, 43] in the literature. It is a syndrome and implies the presence of other congenital anomalies. Genitourinary deformity is invariably present. Other commonly reported anomalies include deformities of the thoracic cage, orthopedic deformities, particularly of the lower extremities, and certain anomalies of development of the intestinal tract.

Less commonly reported anomalies include pulmonary emphysema, patent urachus, accessory breasts, Meckel's diverticulum, abnormal gall bladder position, spinal cord anomalies[29] and inguinal hernia. Our case report adds two other congenital curiosities, that of ectopic liver tissue and a ventral bladder mesentery.

An estimate of the frequency of certain anomalies is gained from previously reported cases. Deficiency of the abdominal muscles is the primary defect designating this syndrome. The lateral flat abdominal muscles are those most often deficient. The rectus abdomini are usually present, but frequently hypoplastic. Less commonly, the deficiency or absence of the quadratus lumborum or latissimus dorsi has been reported. No anomaly of the psoas has yet appeared in the literature. At least one case[52] is reported in which normal muscle development occurred at puberty.

Genitourinary anomalies are present in over 90 per cent of the cases. Hypertrophy and dilatation of the bladder is the most common finding. Ureterectasis, pyelectasis, caliectasis, renal atresia and cryptorchidism are frequent. Less common defects include patent urachus, bladder umbilical fixation, urethral obstruction, bladder neck obstruction, bladder diverticuli, pinhole meatus and intra-abdominal testes.

Various orthopedic deformities are reported. Perhaps the most frequent of these are pigeon breast, Harrison's groove and talipes deformity. Also mentioned have been congenital hip dislocation, spina bifida, head and neck retraction, pectus excavatum, webbed toes and other minor chest wall deformities. One gains the impression that some 30 to 50 per cent of cases possess orthopedic deformities.

According to Silverman,[43] about 20 per cent possess a gastrointestinal anomaly. Most of these involve the mid gut, which gives support to the theory that most deformities in this syndrome are due to maldevelopments during the fifth to tenth week of intrauterine life. Some degree of malrotation is common. Only one case[43] possessed a muscle deficiency and gastrointestinal anomaly alone. A mobile right colon is mentioned several times; however, this should be classified as a minor degree of malrotation. Unusual, or incidental findings, include muteness,[17] abdominal opening of terminal ureters, diastasis recti,[28] pulmonary emphysema and megacolon. We wish to report two additional curiosities; a ventral bladder mesentery and ectopic liver tissue situated within the falciform ligament of the liver.

More than 60 per cent die in early infancy.[6] Obstructive uropathy, urinary infection and respiratory infection are by far the most common causes of death. Early deaths have been reported due to diarrhea, "wasting," "collapse," emphysema, erysipelas, furunculosis, pericarditis, septicemia and intestinal obstruction.

Few patients past the age of puberty have been reported. Physiologic factors to be controlled in this older age group include constipation, abdominal weakness, respiratory infections and urinary stasis. Surgical attack on the syndrome has largely been confined to the correction of intestinal and genitourinary anomalies. Recent authors have recognized the importance of the primary defect (abdominal muscle deficiency) in preventing effective tracheobronchial toilet, urinary stasis and bowel evacuation. Prior to DeBord's surgical repair (1955),[9] treatment of the lax abdomen consisted solely of the use of ab-

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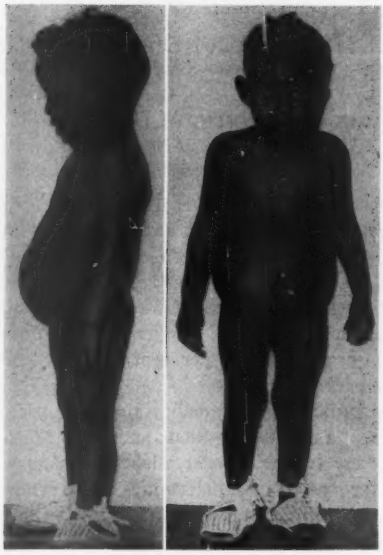

FIG. 1. Negro boy, age 3, with congenital deficiency of the abdominal musculature.

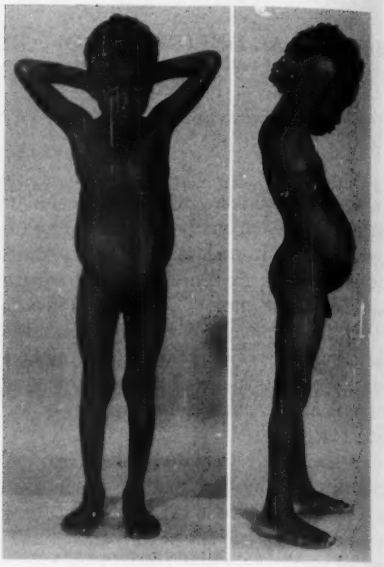

FIG. 2. The patient at age 6, before correction of lax abdomen due to congenital deficiency of abdominal musculature.

dominal binders. We should like to report our method of repairing the abdominal defect in one case.

CASE REPORT

The patient, a Negro boy, was first seen at another institution shortly after birth. There, a diagnosis of congenital deficiency of the abdominal musculature was made and an abdominal binder prescribed.

In October 1956, at the age of six, he was seen at Hermann Hospital and admitted for study and surgical repair.

The past history was not remarkable, except for repeated upper respiratory infections and a persistent cough. The threat of repeated respiratory infections induced us to attempt repair of the lax abdomen. No genitourinary symptoms had ever been present.

Examination showed a six-year-old Negro boy who appeared healthy and alert. He weighed 36 lb. and was 41 in. tall. Complete physical examination revealed normal findings, except for the following: The flat muscles of the abdomen were markedly deficient and the recti hypoplastic. The chest configuration was normal, but upon coughing, almost all expulsive effort was expended upon the flaccid abdomen, causing tussive protrusion of its walls. Bronchial rhonchi changed but did not disappear after coughing. He had bilateral cryptorchidism, a waddling gait and exaggerated lumbar lordosis.

Colon and chest x-rays were normal. Urine culture was sterile. The intravenous pyelogram revealed right ureterectasis, pyelectasis, and calicectasis; the left side was normal. Cystometrogram showed a marked hypotonicity of the bladder; 400 cc. of fluid was instilled with no voiding urge and no pressure rise above 9 cm. of water. Cystograms showed an enormous bladder capacity.

Preoperative physical medicine consultation is summarized: "The rectus abdominis muscles are both weak, markedly so on the right. A point to observe is that many normal children cannot pull up to a sitting position using abdominal muscles until six years of age, so some improvement may be noted later. The abdominal obliques seem to be completely absent. Possibly there are a few functional fibers on the left; the right side exhibits more bulging. The obliques are the supporting muscles; their absence would cause stretching of the recti in time, regardless of their strength. The quadratus lumborum muscles are normal."

Pulmonary function studies showed a vital capacity of 533 cc. (predicted 800 cc.). The maximum breathing capacity was 13.8 L. per min. Impression: a reduction of ventilatory capacity presumably due to poor muscle support.

Surgical repair was effected November 23, 1956.

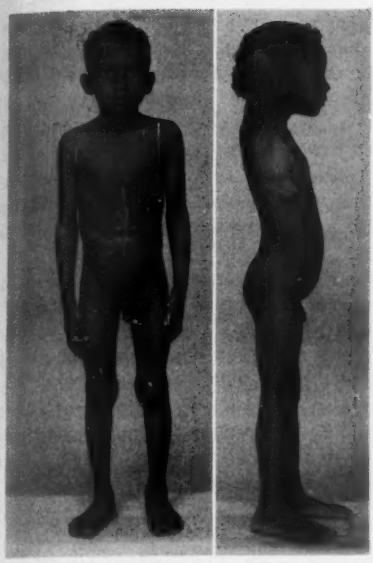

FIG. 3. Four months postoperatively. Note improvement in posture, lumbar lordosis, and protruberant abdomen.

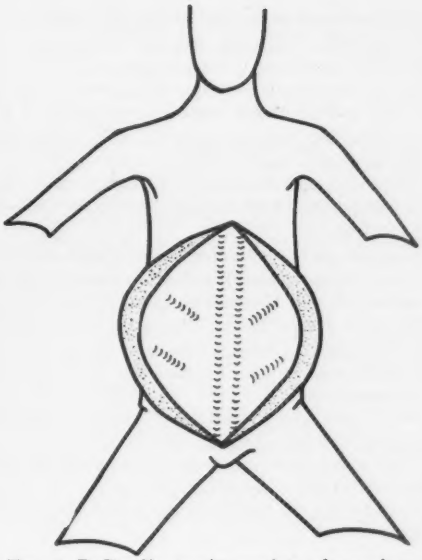

FIG. 4. DeBord's repair consists of a series of extraperitoneal tucks in the fibromuscular layer. (DeBord, 1955.)

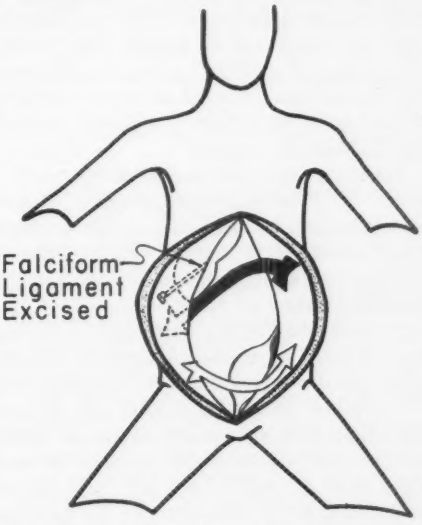

FIG. 5. Method of repair used in the present case. The entire thickness of the fibromuscular layer is overlapped. (Fitch and Denman, 1956.)

The thin skin and subcutaneous layers were reflected from the midline to the midaxillary line, bilaterally. The fibromuscular layer was split in the midline from the xiphoid to the pubis. This layer was thinnest in the upper right quadrant where it approached $\frac{1}{16}$ in. in thickness. Massive overlap of the right flap, superficial to the left, was done after excision of the falciform ligament of the liver. In effect, this transposed the two recti, except at their costal and pubic attachments. Two additional oblique tucks, after the method of DeBord, were taken to imbricate the right upper quadrant. Four perforated No. 14 French catheters were placed in the subcutaneous space and connected to continuous suction to avoid serum accumulation. A stellate piece of excess skin was excised and a cruciate scar resulted.

Additional malformations noted at surgery consisted of a completely unattached ascending colon and distal small bowel mesentery, bilateral abdominal testes with mesenteries and an attachment of the bladder to the umbilicus and abdominal wall by an obliterated urachus and ventral bladder mesentery. The enormous bladder and ureterectasis discovered preoperatively was confirmed.

The postoperative course was benign. Gastric suction was used for three days to prevent distension. Temperature reached 102°F. for two days, 101°F. until the fifth postoperative day, and then fell to his normal level of 99–100°F. thereafter.

Pathology reported a normal appendix and a nodule of liver tissue within the falciform ligament.

Follow-up studies were carried out on a February 11, 1957, admission. Marked improvement in posture was noted; a straightening of the excess lumbar lordosis, less lateral and anterior sagging of the abdomen and some improvement in the waddling gait had been attained. The blood urea nitrogen was 7 mg. per cent.

The urology consultant felt that there was very slight improvement noted in the intravenous pyelogram.

The physiotherapy consultant stated that abdominal support would be indicated later when the abdominal wall becomes progressively stretched. The child should not be allowed to become overweight, because omental fat and fat in the abdominal wall would increase the strain on the abdominal wall.

The orthopedic consultant made no specific diagnosis of the waddling gait.

Pulmonary function study showed a vital capacity of 711 cc. (predicted 800 cc.). The vital capacity approached a normal value since operation.

DISCUSSION

In 1955, DeBord[9] reported the 63rd authenticated case of the syndrome of congenital deficiency of the abdominal musculature (Fletcher's case[12] of omphalocele is rejected and Greene's representation[17] of Daut's case[8] is counted as one). Our case brings the count to 64, not including recent reports by Roberts,[41] Jameson,[27] Hecker[20] and Roviralta.[42]

DeBord's case was the third reported female.[22, 43, 9] Fletcher's case[12] was one of omphalocele in a female infant and is not pertinent. Credit should go to Fröhlich[14] for reporting the first case in 1839. His was one of the few with descended testes.

Approximately 50 of these 64 cases were either dead or under three years of age when reported with reference to their outcome. Two of the total were reported at ages 60 and 70, and obviously must have had very minor degrees of genitourinary and respiratory difficulties. This leaves some 10 or 15 cases in the late childhood and young adult group which deserves the most rigid analysis. This salvagable group should be studied without reference to the ominous outlook implied by the high morbidity and mortality of the perishable infant and young childhood group. However, the published reports of this age group are quite rare.

The genitourinary anomalies have received many varied surgical approaches.[8] We found no mention of surgical or hormonal correction of cryptorchidism in the literature, this is in spite of the emphasis upon the malignant potentialities of an undescended testicle. Many urologists, however, feel that a completely intra-abdominal organ does not carry the same danger of malignant degeneration as does one trapped within the confines of the inguinal canal. However, the exact position of the cryptorchid organ has rarely been stated.

Other surgical attacks have been necessitated by obstructing gastrointestinal anomalies.

With the possible exception of recent reports,[20, 42] only DeBord has previously described a direct attack upon the abdominal laxity. We are in agreement with him that this is most important in improving respiratory function, decreasing respiratory infections and reducing mortality due to respiratory complications.

SUMMARY

The presence of congenitally deficient abdominal musculature implying a syndrome with associated genitourinary, gastrointestinal and bony anomalies is again emphasized.

Separation of reported cases into the rare correctable group, the more common hopelessly malformed group, and the clinically nonsignificant group has been pointed out as a practical approach to better evaluation of statistics.

Infection, obstructive uropathy, and impaired pulmonary function is the triad challenging management.

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GASTROJEJUNOCOLIC FISTULA*

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Gastrojejunocolic fistula is one of the most serious complications resulting from the surgical treatment of peptic ulcers; however, it does not occur often enough for the average general surgeon to become familiar with its diagnosis and treatment. It may occur anytime from a few weeks to years after the initial surgery. Fortunately, gastrojejunocolic fistula is less frequently encountered now than it was a few years ago.

We believe that the less frequent occurrence of gastrojejunocolic fistula today has been the direct result of a more thorough surgical attack upon the peptic ulcer than was practiced 15 to 20 years ago. Berman[1] stated that "this condition (gastrojejunocolic fistula) is a man-made disease," *i.e.*, it follows incomplete surgery for the ulcer diathesis. This may well be true in a majority of cases, but all gastrojejunocolic fistulas are not secondary to surgery. In our series of 8 patients with gastrojejunocolic fistulas at the University of Maryland Hospital, half of the fistulas were preceded by surgical attempts to cure gastric or duodenal ulcers and the other half were due to primary disease.

By far the majority of gastrojejunocolic fistulas reported in the literature are shown to follow operative treatment of peptic ulcers. Operative treatment in most cases was often a method we now deem inadequate, such as gastrojejunostomy without resection. Retrocolic gastrojejunostomies were more prone to gastrojejunocolic fistula formation than were antecolic anastomoses. The consensus of opinion is that gastroenterostomy or a subtotal gastric resection followed by a gastric, marginal or jejunal ulceration may lead to the formation of a gastrojejunocolic fistula. In about 10 per cent of the patients that develop stomal ulceration, a gastrojejunocolic fistula will appear. In the period covered by our study, there were 13 patients with stomal ulcerations and 4 other patients with gastrojejunocolic fistula that followed operations for peptic ulcer. Ransom[5] reported 8 such instances in a series of 47 cases of stomal ulceration. The Mayo and Lahey clinics[2] report an incidence of about 10 per cent.

The largest series of cases of gastrojejunocolic fistula, and one of the most complete studies in the literature, was reported by Lowdon[3] in 1953. Marshall[4] reported an impressive number of cases at a recent meeting of the Southern Surgical Association.

The majority of patients with gastrojejunocolic fistulas have severe diarrhea and weight loss, and about half of them will have fecal vomiting and/or fecal breath. Upon examination the patients are usually emaciated and are often in electrolyte imbalance. Occasionally there is soreness in the upper abdomen, but usually the abdominal examination is negative. Rarely is a mass palpable, and if it is, one should suspect the presence of a neoplasm. The most important single diagnostic aid is the barium enema. When barium can be seen to flow directly into the stomach and jejunum, a positive diagnosis of gastrojejunocolic fistula is made. Upper gastrointestinal tract roentgenograms will often show an ulcer, but in many instances will fail to show the fistula.

The preoperative treatment is most important and should be conducted vigorously. It should include attempts to correct fluid and electrolyte imbalances, anemia, and malnutrition. Antibiotic therapy, both orally and parenterally, is an important adjunct in the preparation of these patients for surgery. Gastrojejunocolic fistula should be treated by operative measures and undue delay should be avoided.

Since the 1930's many operative procedures have been advocated. These procedures have varied from a one-stage dismantling of the fistula, closing the colon and performing another gastroenterostomy, to multistaged operations with or without gastric resection. The present trend is in the direction of a one-stage operation which includes a subtotal gastric resection. By performing a subtotal gastrectomy, one is attacking the ulcer diathesis which appears to be a major factor in the formation of a gastrojejunocolic fistula.

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CASE REPORTS

Case 1. C. F., a 42-year-old white male, was first admitted to the Univerlty of Maryland Hospital in 1939 with a chief complaint of epigastric pain of 12 years' duration. Routine examinations revealed a chronic duodenal ulcer which was confirmed at laparotomy. A posterior gastrojejunostomy was performed, and the patient had an excellent postoperative course. He did quite well until 1946, at which time he returned to this hospital complaining of diarrhea of 6 months' duration with 6 to 12 loose stools daily. Bowel movements, containing undigested food, occurred approximately 15 min. after meals. There was a weight loss of 30 lb. over this 6-month period.

Physical examination revealed a chronically ill patient with obvious weight loss, but otherwise revealed no abnormal findings. Barium enema showed the presence of a gastrojejunocolic fistula. After vigorous preoperative preparation, a laparotomy was performed and the presence of a gastrojejunocolic fistula was confirmed. The fistulous tract was resected and a gastrorrhaphy performed. The jejunum and colon were repaired by respective end-to-end anastomoses. The postoperative course was uneventful. This patient was reviewed in 1949 and again in 1956 and found to be healthy and asymptomatic.

Case 2. E. W., a 41-year-old white male, was admitted to the University of Maryland Hospital in 1943 with complaints of fecal vomiting and diarrhea with 5 to 8 loose stools daily for seven months. Past history revealed that a posterior gastroenterostomy had been performed 13 years before for a peptic ulcer. Physical examination revealed marked emaciation and tenderness in the left upper quadrant of the abdomen. Barium enema revealed a gastrojejunocolic fistula.

At operation, the fistulous tract was resected; the stomach, colon, and jejunum were repaired, and another posterior gastroenterostomy was performed. Postoperatively the patient developed a small bowel obstruction which responded to conservative therapy. His last examination was in 1954 (13 years after resection of the fistula), at which time he was asymptomatic. There was no physical or roentgenologic evidence of peptic ulcer or gastrojejunocolic fistula.

Case 3. J. M., a 36-year-old white male, was admitted to the University of Maryland Hospital in 1942 complaining of diarrhea, weight loss of 20 lb., bloody stools, and fecal vomiting of 4 months' duration. Past history revealed that this patient had had an anterior gastroenterostomy four years previously for a peptic ulcer. Physical examination revealed a markedly emaciated and anemic white male, and a gastrojejunocolic fistula was demonstrated by barium enema.

At operation, the fistula was resected and the stomach opening closed. Continuity of the jejunum and colon was restored by respective end-to-end anastomoses. Postoperatively the patient's temperature rose progressively to 106°F. and a diagnosis of peritonitis was made. There was no response to intensive therapy, and the patient died on the sixth postoperative day. Permission for an autopsy was denied.

Case 4. J. C., a 64-year-old colored male, was first admitted to the University of Maryland Hospital in 1945 complaining of left lower quadrant abdominal pain, indigestion, and vomiting for several weeks. Past history revealed that four years previously he had had a posterior gastroenterostomy and pyloric crush for a duodenal ulcer.

Physical examination revealed marked weight loss and moderate anemia. An upper gastrointestinal series revealed a poorly functioning gastrojejunostomy without evidence of a stomal ulcer. The patient was treated medically and did well for four years.

Upon his second admission in 1950, he complained of epigastric pain, nausea, vomiting, and weight loss of 3 months' duration. Physical examination revealed evidence of weight loss, marked abdominal distention and mild anemia. A barium enema revealed a gastrojejunocolic fistula. Upon laparotomy, the fistulous tract was resected and the stomach and colon openings were closed. Ten centimeters of jejunum were resected, followed by an end-to-end anastomosis and an anterior gastrojejunostomy. Postoperatively the patient developed peritonitis and died on the third postperative day. No autopsy was performed.

Case 5. J. S., a 41-year-old white male, was first admitted to the University of Maryland Hospital in 1939 with a complaint of nausea, diarrhea (five bowel movements daily), and a painful left upper quadrant abdominal mass. The patient was explored and an abscess near the splenic flexure of the colon was found and drained. It was thought by the surgeon that the abscess resulted from a perforated malignant lesion of the colon. The patient did well postoperatively; however, he developed a draining sinus from the incision. One year later he was again admitted for the draining sinus and persistent diarrhea.

Physical examination revealed marked malnutrition and multiple draining abscesses on the anterior abdominal wall. Barium enema revealed a gastrojejunocolic fistula. The only operation performed was incision and drainage of the abscesses. A fecal fistula then developed, but the

patient refused further surgery. We have been unable to trace this patient since his discharge from the hospital.

Case 6. A. H., a 42-year-old colored male, was first admitted to the University of Maryland Hospital in November 1946 complaining of abdominal pain, nausea, vomiting, weight loss, and tarry stools for an undisclosed period of time.

Physical examination was noncontributory except for anemia and malnutrition. An upper gastrointestinal x-ray series revealed a gastric carcinoma. A subtotal gastrectomy and a retrocolic gastrojejunostomy were performed. The postoperative course was satisfactory.

His second admission, four months later, was for a high intestinal obstruction with hematemesis. Upon laparotomy the neoplasm was found to be widespread. There was obstruction at the site of the gastrojejunostomy. An anterior gastrojejunostomy was performed and the postoperative course was uneventful.

The patient was again admitted, three months after his second admission, in a terminal state with marked fluid and electrolyte imbalances. He had vomited all foods and liquids for one week prior to admission. He died the day following admission, and a postmortem examination revealed a carcinoma of the stomach with generalized abdominal metastasis, a gastrojejunocolic fistula, and partial intestinal obstruction.

Case 7. V. W., a 49-year-old white male, was first admitted to the University of Maryland Hospital in April 1946 complaining of nausea, hematemesis, weakness, diarrhea, and melena of 4 days' duration. Past history revealed that the patient had been treated for Addison's disease for several years. Physical examination revealed evidence of weight loss, a moderate anemia, and positive evidence of Addison's disease. A barium enema revealed a lesion of the splenic flexure of the colon and a gastrojejunocolic fistula.

At laparotomy, a gastrojejunocolic fistula was found and a resection of the greater curvature of the stomach and a splenectomy were performed. The splenic flexure of the colon and 10 cm. of jejunum were resected and restored respectively by end-to-end anastomoses. The pathologic report was papillary adenocarcinoma of the colon and acute and chronic inflammation of the stomach and colon.

Postoperatively the patient did well for the first three days; however, on the fourth day, he became hyperpyrexic and paraplegic. A neurosurgical consultation was obtained and an exploratory laminectomy was performed with a tenative diagnosis of extradural abscess. There were no pathologic findings upon laminectomy and the patient went rapidly downhill and died on the second day. A postmortem examination was not granted.

Case 8. J. L., a 31-year-old colored male, was admitted to the University of Maryland Hospital in 1949 complaining of "stomach ulcers."

Past history revealed that a tuberculous cervical node had been excised in 1934, followed by irradiation therapy. Three months prior to admission, the patient had first complained of postprandial epigastric pain relieved by vomiting. It was also noted that he passed undigested food *per rectum* about 30 min. after eating. The patient lost 20 lb. in 3 months. No history of previous operation for peptic ulcer was found. Upper gastrointestinal series performed four weeks prior to admission revealed a partial pyloric obstruction. The patient was treated medically and upon admission to the hospital, repeat roentgenograms showed a duodenal ulcer and a gastrojejunocolic fistula without pyloric obstruction.

At laparotomy, the roentgenographic findings were confirmed. An ileosigmoidostomy, as the first stage of a two-stage resection, was performed. In addition, a lymph node and segment of ileum were biopsied. The pathologic report was granulomatous tissue; tuberculosis could not be ruled out. Cultures for tuberculosis were negative. The postoperative course was completely uneventful.

This patient was subsequently followed as an inmate in one of the State penal institutions where he developed pulmonary tuberculosis which cleared with treatment. When seen in 1955 he was asymptomatic and a barium enema at that time failed to show a gastrojejunocolic fistula. Because of the apparent healing of the fistula, no further operation is contemplated.

DISCUSSION

It has been interesting for us to review the cases of these eight patients with gastrojejunocolic fistulas and to recall the many changes in our concept of the treatment of this problem. Four patients (Cases 1–4) developed this complication as the result of an attempt to cure their peptic ulcer. The methods of treatment, each by a different surgeon, left much to be desired according to our present concepts of the surgical treatment of peptic ulcers.

Cases 5, 6, and 7 represent the formation of gastrojejunocolic fistulae secondary to malignant diseases of the gastrointestinal tract.

To attribute a gastrojejunocolic fistula to tuberculosis is an attractive and interesting thesis in the eighth case. It gains some support

from the history of glandular tuberculosis and the development of pulmonary tuberculosis in this patient. More support is attained by the finding that the gastrojejunocolic fistula healed following ileosigmoidostomy and treatment with antituberculous drugs. The pathologic report of the biopsy was noncommital, and complete proof of the diagnosis is lacking.

In general, one is impressed by the varieties of treatment of gastrojejunocolic fistulas. Patients with this affliction require a well planned period of preparation before being subjected to the magnitude of a one-stage operative procedure that would combine resection of a gastrojejunocolic fistula with a subtotal gastrectomy. With the present-day improved adjuncts to surgical treatment, various authors are now reporting greatly lessened morbidity and mortality in treating gastrojejunocolic fistulas using a one-stage resection. In some instances, however, a multistaged procedure may be mandatory.

SUMMARY

1. A brief discussion of gastrojejunocolic fistula is presented.

2. Eight cases of gastrojejunocolic fistulae are presented.

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DISSECTING ANEURYSM OF THE AORTA: A NEW SURGICAL PROBLEM*

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In past years, the antemortem diagnosis of dissecting aneurysm of the aorta was considered a rare and brilliant achievement, and many clinicians were little concerned that the diagnostic accuracy in this condition was only 10 per cent. Recent advances in surgical treatment of this disease, however, have changed dissecting aneurysm from a "beautiful case for the Clinical Pathologic Conference" to a more practical problem, and, if early diagnosis is made and treatment instituted, there should be an excellent opportunity to lower the previous mortality rate of 80 to 90 per cent.

We have reviewed a group of 13 autopsied cases of dissecting aneurysm from Memphis hospitals, and they form the basis of this study.

CLINICAL FEATURES OF DISSECTING ANEURYSM

The ages of 3 patients in our study were in the fourth decade, 7 were in the fifth decade, 2 in the sixth, and 1 in the seventh. There were 10 men and 3 women in the group, 8 of whom were white and 5 were colored.

Ten of the patients had significant histories of hypertension, 1 had previously had a myocardial infarction, 1 had a history of angina pectoris, and 3 had prior renal disease.

The chief complaint in 7 of the 13 patients was chest and/or back pain. In 3, the chief complaint was abdominal pain, and 1 each had neck and jaw pain, flank pain, and headache and dyspnea.

The duration of these complaints, in 5 patients, was less than 8 hr., and in 3 patients more than 1 day but less than a week. In 4 patients, the duration was more than 1 week, and the duration of the attack of 1 individual was unknown as he was found dead in bed.

The pain was severe in almost each instance and could not be relieved by narcotics. In 7 patients, the pain began in the chest and radiated into the back, whereas in 3, it began in the epigastrium and radiated into the flanks. In 2 others, the pain was first noticed in the low abdomen and progressed to the lower extremities. In the latter 2, the legs had evidence of arterial occlusion. Neurologic symptoms were present, manifested by a brief period of stupor and semiconsciousness in 1 patient, and another had a brief convulsion. Severe headaches were present in 2 patients, and 2 others had dyspnea.

Findings were quite varied on physical examination. On admission examination, in 10 patients, the blood pressure was elevated to hypertensive levels. The predominant physical findings are noted below:

Findings:	*No. of Patients:*
Signs of shock	4
Apical systolic murmurs	5
Pulmonary rales	3
Abdominal tenderness	3
Absent pulses in lower extremity (Signs of arterial occlusion)	2
Enlarged liver	2
Basal diastolic murmur	1
Blood pressure difference in extremities	1
Signs of pleural effusion	1

The predominant roentgen findings are as noted in the following:

Findings:	*No. of Patients:*
Enlarged heart	7
Increased size of aortic shadow	5*
Bilateral pulmonary edema	2
Enlarged pulmonary conus	1
(X-ray not carried out)	4

* In one patient, the admission x-ray showed a normal sized aorta, but subsequent films showed marked aortic enlargement.

The laboratory findings showed a low hemoglobin, low red blood cell level, and low hematocrit in 4 patients. An elevated blood amylase in 1 patient led to an erroneous diagnosis of acute pancreatitis. Nonprotein nitrogen levels were elevated in all patients in which it was determined.

The electrocardiogram showed evidence of coronary disease with possible infarction in 2

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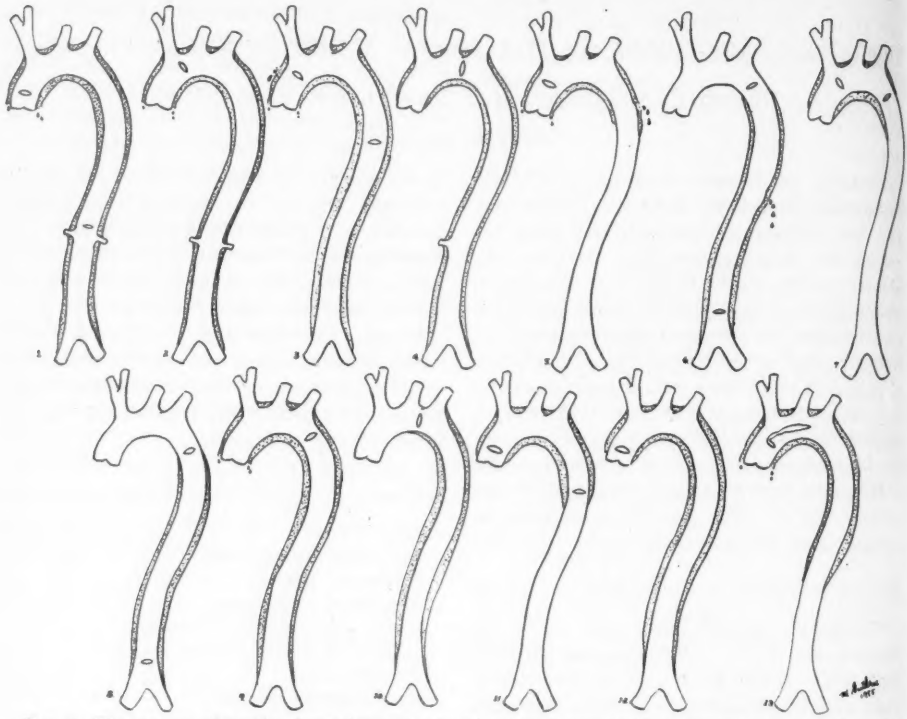

FIG. 1. The extent of aortic dissection and site of intimal tear in 13 autopsied cases of dissecting anurysem of the aorta.

patients. In 5 patients, it showed only findings of left heart strain and hypertrophy with no evidence of infarction. The electrocardiogram was not done in 6 patients.

The time interval from the onset of significant trouble until death is noted below:

Time:	*No. of Patients:*
12 hr.	1
24 hr.	3
2 days	1
More than 2 days but less than 7	3
More than 1 week	5

The primary cause of death in each patient was dissecting aneurysm, with cardiac tamponade found in 3 of these patients, hemorrhage and shock in 9 others, and apparently congestive failure in 1.

PATHOLOGY

Of prime importance to one who would recognize and surgically treat dissecting aneurysm is the gross appearance of the lesion. This was described as hemorrhagic and markedly distended in 5 patients; in 1 patient, the aorta measured 8 cm. in diameter. In those in which rupture had occurred, a large mass of blood clot was present in that area.

The locations of dissection were as noted in figure 1. In 6 patients, dissection was from the aortic valve to the aortic bifurcation. In 4, it involved the arch of the aorta from the valve to the upper thoracic aorta, and in 3 patients, it primarily was from the left subclavian area to the aortic bifurcation. In all instances, an intimal split was noted (fig. 1). This was either just above the aortic valve or in the arch area in 10 patients, and was just distal to the left subclavian in the remaining 3.

Dissection extended into the arch branches in 2 patients, into renal vessels in 1, and into the iliac vessels in 2. This caused partial or nearly complete obstruction of these branches in all patients. Rupture of the aorta occurred in 9 patients. This

rupture was into the pericardium in 6, into the pleural space in 2, and into the pericardium and pleural space in 1 only.

Evidence of spontaneous re-entry of the dissection was present in 5. These re-entry points were in the thoracic aorta in 2 patients, in the abdominal aorta in 2, and into the iliac artery in 1. In 2 of the above, a well formed double-barreled aorta was present.

Microscopic examination revealed evidence of so-called idiopathic medionecrosis in 12 of the 13 patients. In 10, arteriosclerosis was present in a moderate to a marked degree. Evidence of syphilitic aortitis was present in 1 patient only.

Significant associated pathology consisted of arteriosclerosis in coronary and other vessels in 8 patients, pulmonary tuberculosis in 1, pulmonary infarction in 1, and cardiac enlargement with hypertrophy recorded in 11.

DISCUSSION

A review of the literature in 1950 by Levinson, Edmeades, and Griffith[7] revealed that of 734 reported cases, only 10 per cent had a correct antemortem diagnosis. In their own group of 58 patients, the incidence of correct antemortem diagnosis was 27.5 per cent. With the advent of surgical treatment of this disease, it is mandatory that the percentage be substantially improved.

In general, the literature is in agreement that the incidence is most frequent in the fifth, sixth, and seventh decades of life, and men predominate in incidence about 2.5 to 1. Schnitker and Bayer,[10] however, found that 24 per cent of their cases were in individuals under 40 years of age.

As the aorta supplies all parts of the body, the symptoms that serious aortic disease produces can be multiple and quite varied, but the outstanding symptom is pain of a severe nature. Twelve of the 13 patients in this series had outstanding complaints of acute pain. If the thoracic aorta is the original site of dissection, the pain is felt in the chest and upper back. This pain may go into the neck, head, jaw, or arms, if the innominate, carotid or subclavian vessels are involved. As dissection progresses, the pain moves down the back and chest to the abdomen or low back, and may radiate into the legs. Pain was substernal and similar to that of coronary occlusion in only 6 per cent of the patients in Logue's[8] series.

The onset of pain is usually sudden, and is not necessarily related to physical exercise. It may be described as tearing, ripping, knifelike, burning, or in general, very severe, and incompletely relieved by narcotics.

In some cases, an episode of syncope may be the initial symptom. Moersch and Sayre[9] have emphasized the frequency of neurologic symptoms and signs in this disease.

According to Levinson and associates,[7] the most common secondary symptoms are dyspnea, vomiting, orthopnea, and oliguria. The physical signs of pallor, cold clammy skin, elevated pulse, and dyspnea are the most common. One should always check for signs of arterial occlusion in the distal arteries.

The presence of elevated blood pressure in the upper extremities, with low blood pressure in the lower extremities is very suggestive, as is the presence of hypertension with signs of shock. The presence of an aortic diastolic murmur is suggestive and was noted in 56 per cent of the cases reported by David and co-workers.[4] This was noted in only 1 of our patients, however. Other miscellaneous findings may be pulsating abdominal mass or signs of chest fluid or cardiac tamponade.

The routine blood studies may reveal anemia consistent with blood loss. Nonprotein nitrogen was consistently elevated in all of our patients in which it was determined. Urinalysis may show red cells or albumin.

The electrocardiogram is often a stumbling block and can be a tip-off to diagnosis. In dissecting aneurysm, it is the exception for the electrocardiogram to show findings typical of coronary occlusion. The most common pattern is that of left heart hypertrophy and strain (Levinson: 62 per cent[7]). In David's series of 17 patients, there were no normal electrocardiograms, but neither were there any typical of the changes of infarction.

The chest roentgenogram, in about one-half of the patients, showed a widened aortic shadow,[2] and enlargement of the aortic size demonstrated in serial films was most suggestive.

The use of angiocardiograph is most helpful, according to DeBakey, Cooley, and Creech.[6] They state that characteristically the dye is heavily concentrated in the true aortic lumen, whereas the false passage appears as a lateral or medial superimposed channel.

The differential diagnosis usually lies between coronary occlusion, ruptured aneurysm, mesen-

teric thrombosis, acute pancreatitis, and pulmonary embolism. Blain[7] has pointed out the importance of distinguishing between dissecting aneurysms and acute abdominal disease, although his conclusions that one should avoid surgery in dissecting aneurysm must now be altered.

One of our patients was treated as a case of acute pancreatitis, and another as a case of femoral artery embolism.

TREATMENT

Gurin, Bulmer, and Derby,[5] in 1935, were apparently the first to surgically treat a dissecting aneurysm. Their approach was to open the iliac artery, excise a portion of the internal wall of the aneurysm, and allow the artery to bleed back into its true lumen. This restored circulation in the occluded iliac artery, but the patient died of uremia on the seventh postoperative day.

Johns[6] described, in 1953, a case in which he closed a rupture of a dissecting aneurysm by suturing. This patient also died of renal failure.

Shaw[11] recently described a patient in whom a fenestration was made between the true and false lumens in the area of the abdominal aorta. This reestablished the distal blood flow. A new point in technique was that he closed by suture the plane of the dissecting aneurysm below the aortotomy. This patient also, unfortunately, died of uremia.

Cooley, DeBakey, and Creech[3] have now operated on 13 patients with dissecting aneurysm and there have been 10 survivors. They have used a fenestration type of operation in the classical dissecting aneurysm, excision with grafting in those in which dissection begins below the left carotid, and lateral aortorrhaphy in at least 1 case. The details of their methods are very well recorded in the recent literature.

Warren, Beckwith, and Muller[13] have reported 4 patients operated upon, with 1 survival.

Swann and Bradsher[12] reported a patient who was successfully operated on by division of the aorta and production of a re-entry site for the dissection.

From the small number of surgical cases recorded, one can readily discern that this is not only a new treatment, but one which has not been fully developed. It appears at this point to be a tremendous improvement over the watch, wait, and hope methods which result in fatality in 90 per cent of such patients.

SUMMARY

1. Thirteen cases of dissecting aortic aneurysm have been studied and their clinico-pathologic features described.

2. With the advent of a successful surgical treatment, it behooves both surgeon and internist to improve their diagnostic acumen in regard to this condition.

3. One should consider dissecting aneurysm in any patient with severe chest, back, or abdominal pain. Methodical study with proper use of laboratory aids should enable one to diagnose these patients in 80 to 90 per cent of instances.

4. Dissecting aneurysm is a surgical emergency, and the thoracic surgeon should lose no time in carrying out treatment.

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SPONTANEOUS RUPTURE OF THE ESOPHAGUS*

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Spontaneous rupture of the esophagus has been reported in less than 200 patients, of whom less than 50 have survived. In order to extend the clinical knowledge of this disease, to reemphasize the importance of the early diagnosis, and to summarize the treatment of the late case, we are presenting a current review of the problem in reference to 7 patients who did not have the benefit of early surgery.

The exact frequency of the catastrophe is unknown, since no accurate reports of its incidence have appeared in the literature. Although the first reported case was in 1724,[3, 24] there were only 4 premortem diagnoses made prior to 1936.[26] By 1943 there had been 40 cases reported in the literature, with 3 survivals following conservative therapy.[3] In the next 13 years the total number of reported cases had risen to 157 with 35 survivals.[6] The total incidence at Memphis Veterans Administration (Kennedy) Hospital, Birmingham Veterans Administration Hospital and University Hospital in Birmingham is about 1 patient per 24,000 admissions. The true incidence of the disease is clouded by the indefinite classification, with many cases included in the literature which were due to preexisting disease.

The disease is most common in patients from 35 to 55 years of age,[13] but may occur at any age.[6, 13] Our cases occurred in males from 40 to 63 years old, with a mean of 53 years.

This affliction occurs most frequently in the male; about 85 per cent of the cases are in this sex.[5, 13, 14] There are only a few Negroes among the reported cases. One of the 4 patients from Kennedy Hospital was a Negro; about 28.5 per cent of the persons admitted here are colored. At University Hospital (Birmingham) about 45 per cent of patients admitted are Negroes, yet no cases have been observed there among the colored patients.

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ETIOLOGY

Perforations of the esophagus may be the result of: instrumentation, penetrating trauma, neoplasia, congenital anomalies, esophagitis, "peptic" esophageal ulcers, increased esophageal pressure from air or contrast media, infection, strictures, chemicals, blunt injury to the abdomen, central nervous system diseases,[7, 8] erosion of aneurysms and increased intra-abdominal pressure due to coughing,[15] defecation,[3] asthma,[18] or, most commonly, vomiting.[2, 17, 20, 23, 25, 26] Gastric acid and enzymes may play a part in the etiology, and gastric glands may be found in the lower third of the esophagus. Spontaneous rupture should be defined as perforation of the normal esophagus resulting from mechanical forces entirely within the patient's own body and divorced from external violence. With such strict criteria for diagnosis, it immediately becomes apparent that a great many of the reported cases were not true spontaneous ruptures.

The disease is frequently associated with poor nutrition, upper gastrointestinal disease,[1, 4, 10, 14, 20, 23, 26] chronic or severe illness,[1, 20, 26] alcoholism and brain disease.[1, 7, 8, 22] Frequently, the patient is in the postoperative period following intra-abdominal surgery.[2, 6, 23] The disease frequently occurs in hospitalized patients,[5, 23] as in Case 6. This frequency of association with other diseases has so impressed many investigators that some believe that spontaneous rupture of the esophagus never occurs in the absence of preexisting disease.[1, 25] The more careful the study of the reported cases, the more this is noted; yet, there remain patients in whom the lesion has been visualized and studied grossly and microscopically, and in whom the history is so clear that the authors are convinced that such an entity exists.

Although the diagnosis of spontaneous rupture may not be proved in an individual case, the symptom complex of this disease and that of

spontaneous perforation due to nonmalignant preexisting disease is so indistinguishable that the diagnosis and initial treatment may, for all practical purposes, be considered identical and the strict division somewhat academic.

CLINICAL PICTURE

The classic and most common history is that of a previously healthy person having an episode of increased intra-abdominal pressure, usually vomiting or retching, followed immediately by severe upper abdominal and thoracic pain with collapse, dyspnea and shock. Collapse does not respond to shock therapy. Many patients with perforation will not have this typical history. There have been cases reported in which there was no vomiting preceding the pain, and this occurred in 5 of our 7 patients. Shock may be absent, especially early, as in Cases 2, 3, 4, 6, and 7. The vomitus may contain blood. The onset may be insidious with mild epigastric pain, small hematemesis, or subcutaneous emphysema which may cause the patient to seek medical attention.

The pain is typically sudden, severe, unrelieved by analgesics and first located in the epigastrium, followed by substernal and posterior lower thoracic pain. The degree of pain may vary from absent to extremely excruciating and unremitting. Four of our patients had no severe pain. If present, it is almost always constant or progressing. Cyanosis was marked in 2 of our cases, compared with about 60 per cent of the cases studied from the literature.

Physical examination usually discloses shock, dyspnea, cyanosis, subcutaneous emphysema as a late feature and pneumo- or hydropneumothorax, especially on the left. The patient frequently sits up, bent over, or lies on the side with legs and back flexed. Movement causes marked aggravation of the excruciating pain. Subcutaneous emphysema was noted in only 1 of our patients during the first 24 hr., but in 3 patients during the course of the disease. In rare instances this is the first sign of the disease, usually involving the face and neck. Subcutaneous emphysema is reported in about 60 per cent of the cases in the literature.[8, 14, 21] Mediastinal crunch or change in the voice may be present.[6, 25] Abdominal rigidity is common, frequently limited to the epigastrium.[21] There may be slight-to-moderate epigastric tenderness, and this was present in all but 1 of our patients. Dyspnea was present in all of our patients and is one of the most common symptoms.

X-ray of the chest typically shows hydropneumothorax (74 per cent),[22] and mediastinal and, particularly, retrocardiac emphysema, frequently with various other nonspecific effusions and emphysemas. The early film may show only basal atelectasis, mild effusions, widening of the mediastinum or other indeterminate features. It is rarely completely negative (9 per cent),[2] and infradiaphragmatic air is not a feature of the emphysema.

The other usual laboratory examinations and electrocardiograms are typically normal except for an increase of leukocytes and left shift. Temperature may be subnormal early, but soon rises.

While these severe signs are usually present, they are not at all necessary for the diagnosis. However, most of the marked symptoms appear within a few hours.

DIFFERENTIAL DIAGNOSIS

Because of the more frequent history of peptic ulcer and because of the initial abdominal pain and rigidity with circulatory collapse, perforated peptic ulcer is the most common misdiagnosis.[14] Coronary occlusion, dissecting aortic aneurysm, mesenteric thrombosis and spontaneous hemopneumothorax should all be considered. Mediastinal emphysema may be seen in uterine, gastric or duodenal rupture or it may be seen spontaneously.[8] The disease has been misdiagnosed as almost any of the acute abdominal or thoracic diseases. The most important single fact leading to a correct diagnosis is to *think* of the possibility. Spontaneous rupture is one of the diseases in which an immediate diagnosis is necessary to save the patient's life. After history and physical examinations are done, definite proof of the diagnosis is mandatory. If examination has shown abnormal chest findings, the patient may be given 20 cc. of iodized oil to swallow just before the emergency chest x-ray is made. If he is too uncooperative or comatose, the oil may be injected through a Levin tube. This procedure will usually allow a definite diagnosis, including the site of perforation, while the intrathoracic pathology is elucidated. In the patient in whom there is obvious pleural effusion, particularly with respiratory embarrassment, it may be desirable to do a thoracentesis even before a chest

x-ray is made. In this event, and in those patients who already have intercostal tube drainage, a little methylene blue may be given by mouth and promptly recovered from the chest fluid. Another simple ward test includes the testing of the fluid by litmus paper to be sure of its gastric origin. These elementary and very rapid procedures are all that are needed for an accurate diagnosis. They are so easy and inexpensive that they may be performed every time the possibility of a ruptured esophagus passes through the mind of the examiner in a case of obscure acute upper abdominal or thoracic disease. One should use all three tests, because the defect has sometimes not been visualized on a single film of a lipiodol swallow and the dye may not immediately appear in a longstanding case with loculated empyema pockets (Case 3).

PATHOLOGY

The gross appearance of the esophageal lesion is characteristic. There is a linear longitudinal tear, usually on the left posterolateral surface, almost always just above the diaphragm. There are 2 reported cases of transverse tears,[6, 12] 1 of a tear in the middle esophagus[26] and 1 in the cervical esophagus.[24] The mediastinal pleura may be intact, in which instance, there is leakage of gastric contents into the mediastinum only, usually with emphysema which frequently rises to the neck. One or both pleuras may be perforated, with resulting hydropneumothorax. Almost immediately, there is an acute progressive inflammatory process in the adjacent esophagus, mediastinum and pleural cavities. There may be superficial mucosal tears near the point of rupture. The esophagus shows no characteristic microscopic lesion. The immediate inflammatory reaction rapidly progresses to a mixed suppurative mediastinitis and/or empyema. The profound shock usually seen is not easily explained. Various theories have been advanced such as irritation by the gastric contents, pressure interference with great vessel and cardiac mechanics, etc., yet none seems to completely explain the shock. The authors recently observed a patient who perforated the esophagus into a small, six-month-old empyema space remaining after a pneumonectomy, and well drained with a tube. This patient's mediastinum was well fixed so that flexibility and resultant mechanical factors were nonexistent. The pathologic changes included a simple postneumonectomy empyema pocket and some nodes in the mediastinum and around the pancreas with metastatic carcinoma from the old lung lesion. There was a 5-mm. defect from the esophagus into the empyema pocket. However, this patient manifested the sudden onset of severe pain, shock, sweating, apprehension, and, in general, the acute appearance (leading to unexplained death in three days) one associates with a spontaneous perforation with its drastic mediastinal and pleural pathology.

PROGNOSIS

The prognosis of this disease is extremely poor. Without definitive treatment 65 per cent of the patients die within 24 hr., 89 per cent within 48 hr., and almost 100 per cent within 96 hr.[6] Rarely, a patient will live for several days without treatment and can be salvaged by conservative therapy. Treatment, when early and bold, can bring the mortality down to a respectable level.

If the patient is seen within 48 hr. of onset, as soon as the diagnosis is made, the operating room should be alerted for major thoracotomy. These patients are extremely poor risks and one is very tempted to delay surgery until resuscitation and a better general condition are obtained. This is usually a vain endeavor and time spent in its attempted accomplishment is usually wasted, as the patient's condition grows steadily worse. Surprisingly enough, these patients usually improve markedly during surgery. While preparations are being made for surgery, tubes can be inserted into the involved chest for decompression of any tension pneumothorax or removal of fluid. Oxygen is given. A Levin tube is inserted into the stomach and another into the upper esophagus and connected to suction to help prevent further spillage. If there are signs of mediastinal compression, a small suprasternal incision can be made under local anesthesia with finger dissection into the mediastinum and the insertion of a catheter. Fluids are started intravenously (usually whole blood is not indicated in spite of the shock) and massive antibiotic therapy is instituted. Sedation is usually ineffective and may be dangerous.

At surgery, a standard posterolateral thoracotomy incision is used, resecting a lower rib and approaching through the involved side, or left side, if both pleural cavities are involved. The

mediastinal pleura is widely incised and the esophageal defect closed with two layers of nonabsorbable suture. Large tubes are placed in the apex and base of the chest and connected to underwater drainage. The wound is closed in anatomic layers, leaving the mediastinum open.

If the patient has survived several days when he is first seen, surgery is probably best delayed, or possibly avoided.[8–11, 19] Many patients have recovered without repair of the defect, or with dalayed repair.[16, 25] The good results which may follow a conservative approach are not usually appreciated, although the first few cures of this disease ever reported followed conservative therapy.[3] Overholt had a patient, first hospitalized eight days after a 7-cm. rupture, who recovered with conservative therapy and had an apparently normal esophagus thereafter.[1] One series of 5 cases has been treated conservatively with 100 per cent survivals.[1]

Levin tubes are inserted to prevent further contamination, all oral intake is stopped, and intravenous fluids are given. Broad spectrum antibiotics are given and empyema pockets and suppurative mediastinitis are drained by tubes. The patient usually responds to this type of conservative therapy. However, after an initial improvement, a plateau may be reached when closure of the perforation is indicated before any further improvement is likely. This operation is identical to that for the early stages except that it is more difficult and usually necessitates pulmonary decortication. Postoperative breakdown of the esophageal repair is not necessarily an indication for reoperation because these fistulas will frequently close spontaneously. Empyema pockets, mediastinal abscess and suppurative pericarditis (see Case 5) are late complications which are treated in the usual manner. The arduous but rewarding nature of this conservative course is well illustrated by Cases 3 and 5.

CASE REPORTS

Case 1 (Veterans Administration Hospital, Birmingham, Alabama)

History. This 40-year-old white male, during an episode of coughing on January 25, 1954, suddenly developed acute epigastric pain followed by gross hematemesis. He was seen by a local physician who made a diagnosis of perforated peptic ulcer in view of a history of known duodenal ulcer of 2-years' duration. Immediate abdominal exploration was said to have shown no abnormality, although the patient developed shock and cyanosis, which was assumed to have been the result of a coronary thrombosis during surgery. He was given nasopharyngeal oxygen and intravenous fluids. The pulse remained around 120 associated with a high fever, and there was persistent nonproductive cough and development of a left pleural friction rub. About 6 days after surgery the abdominal incision sutures were removed and he was transported by ambulance for over a 100 miles to this hospital.

Physical examination. Blood pressure was 118/80, pulse rate 102 and temperature 98.8°F. Abnormalities were limited to: marked cervical and upper thoracic subcutaneous emphysema, tracheal deviation to the right, and hyperresonance over the left chest with decreased breath sounds. The heart sounds were too distant and quiet to be evaluated. The abdomen revealed a recent upper abdominal surgical incision with complete disruption and evisceration of colon, small bowel and omentum, with cold, dry, cyanotic viscera. Rectal examination was normal.

Laboratory data. Urinalysis was within normal limits. White blood cell count was 25,000 with 78 per cent neutrophils, 6 lymphocytes, 16 monocytes; red blood cell count was 6.0 million; 16 gm. hemoglobin. Blood chemistries were normal. X-ray of chest showed loculated hydropneumothorax on the left with many fluid levels. There was marked mediastinal displacement to the right. Electrocardiogram showed sinus tachycardia.

Tentative diagnosis. (1) Tension pneumothorax with severe mediastinitis, probably secondary to perforated esophagus; (2) evisceration of abdominal incision.

Course. The patient was immediately carried to the operating room. A tube was placed in the left ninth intercostal space at the posterior axillary line and connected to underwater drainage. Intravenous Pentothal anesthesia was given with endotracheal oxygen, and the abdominal incision closed. Following surgery, a tube was placed in the second anterior intercostal space in the midclavicular line, and connected to underwater drainage. The patient remained disoriented, hyperactive, hyperthermic and with marked hyperpnea in spite of nasopharyngeal oxygen and antibiotics. The temperature spiked to 105°F. while the patient was on penicillin, oxytetracycline and streptomycin. Tracheotomy was done to maintain bronchopulmonary toilet and to reduce dead space. The pulse remained at 120 with respirations of 40 and temperature of 105°F. Intravenous fluids and electrolytes were continued, but the patient died on the seventh hospital day, 13 days after onset of illness.

Autopsy. Autopsy revealed a 1-cm. rupture of

the left lateral esophagus, 1 cm. above the hiatus, with severe suppurative mediastinitis and empyema. There was no evidence of previous esophageal or gastric disease.

Case 2 (University Hospital, Birmingham, Alabama)

History. This 53-year-old white male developed sudden severe left anterior chest pain while standing and talking with a friend. The pain was steady and did not radiate. He felt nauseated *after* the pain started, but did not vomit. He was given Demerol, Digifolin and Coramine and sent to the hospital, 2 hr. after onset.

This patient was a known hypertensive for 26 years and had been on a low salt diet for treatment of his hypertension for the past 3 years. Six months previously he had pneumonia which responded well to antibiotics.

Physical examination. Physical examination on admission revealed the following abnormalities: systolic blood pressure 110, diastolic unobtainable; pulse 150; respiration 48; temperature 102°F. rectally. The patient was cold, clammy and moist; however, he was alert and cooperative. The lips were cyanotic. Breath sounds were markedly decreased over the left chest below the fourth rib posteriorly and anteriorly. There was a supracondylar amputation of the left leg.

Tentative diagnosis. (1) Arteriosclerotic and hypertensive heart disease with myocardial infarction; (2) spontaneous pneumothorax.

Course. While in the emergency room the patient received nitroglycerine, morphine, oxygen, atropine, Cedanilid and norepinephrine. Upon admission to the ward his blood pressure was 110/70, then gradually became unobtainable about 1 hr. later.

Extreme dyspnea continued. Left thoracentesis yielded 3,000 cc. of foul-smelling, light brown, turbid fluid. Methylene blue injected through the thoracentesis needle was present in the patient's sputum in 2 min. Nine hours after admission to the hospital the patient's respiration abruptly slowed to 12 per minute and 5 min. later the heart beat and respirations ceased. At this time the patient's blood pressure had not been obtainable for the past 4½ hr. The patient died 11 hr. after onset.

Autopsy. The significant findings were: the left lung was collapsed with bilateral effusions and undigested food in the right pleura; there was a linear tear of about 2 cm. in the lower thoracic esophagus, communicating with both pleural cavities; there was no evidence of previous esophageal disease.

Case 3 (Veterans Administration Hospital, Memphis, Tennessee)

History. This 46-year-old colored male was admitted January 11, 1956, with a chief complaint of epigastric and left lower chest pain of four days' duration. He stated that he was apparently in his usual perfect health until he developed nausea and vomiting after eating "bad food" four days prior to admission. Shortly after this, he developed constant "heavy ache" in the epigastrium with pain radiating around the left lower rib cage to the left costovertebral angle. The pain was aggravated on deep breathing. He denied all other respiratory or gastrointestinal symptoms. He admitted large alcohol intake two weeks prior to onset.

Physical examination. The temperature was 101.8°F., pulse rate 160, blood pressure 108/80, and respirations 40. The patient was slightly cyanotic and appeared acutely ill, in respiratory distress, and lying curled up on the right side. Physical examination revealed the following: there was minimal excursion of the chest during respiration with some inspiratory retraction in the lower right base posteriorly; the mediastinum was slightly shifted toward the right with dullness and decreased breath sounds in the lower half of the right chest; moist rales were heard in the right upper chest and in both posterior lung fields; there was minimal tenderness in the left upper quadrant of the abdomen.

Laboratory data. Routine blood counts and hematocrit were normal. Urinalysis showed +1 albumin, 10–12 white blood cells, 2–4 granular casts. X-ray of the chest revealed multiple cystic areas with fluid levels in both lung fields with questionable effusion on the right.

Tentative diagnosis. Multiple infected cysts of lung with pleural effusion.

Course. Thoracentesis yielded 600 cc. of milky fluid with a fecal odor. This fluid had a cell count of 6,600 white blood cells; 100 per cent polymorphonuclear leukocytes; Gram stain showed Gram-negative rods and Gram-positive cocci.

Penicillin, Gantrisin, Terramycin and nasopharyngeal oxygen were administered. Within 24 hr. he became more dyspneic, with a pulse rate of 150, and examination showed reaccumulation of effusion. Repeat thoracentesis yielded 500 cc. of purulent fluid. A thoracostomy tube was immediately placed in the left pleural space with evacuation of about 1,000 cc. of thick yellow pus. There was marked decrease of dyspnea and the patient's mental condition improved dramatically. Esophageal rupture was considered likely and methylene blue was given by mouth, but no dye was recovered in the drainage. Right pleural

effusion appeared and a tube was placed in this pleural cavity. The pulse was irregular and an electrocardiogram revealed auricular fibrillation. Fluid and electrolyte balance was maintained with intravenous fluids. The patient improved considerably and chest x-ray showed marked improvement on the right, but there was reaccumulation of fluid on the left and a large mediastinal air-fluid level. Another tube was placed in the left chest, but repeat pericardial aspirations yielded no fluid. Methylene blue was again given by mouth four days after onset and immediately appeared in the left chest drainage. A Levin tube was placed in the stomach and another in the midesophagus and suction applied to both. Temperature became normal on the 6th day. By the 13th day there was marked improvement in his condition and x-ray appearance. However, his weight steadily declined and a low grade fever recurred.

On the 23rd day a Lipiodol swallow showed the contrast media to escape from the lower esophagus into both hemithoraces. Cultures of the chest fluid grew a wide mixture of organisms, all sensitive to chloramphenicol, which was then started.

On the 49th day the left chest was explored under endotracheal cyclopropane anesthesia. There were several small pockets of pus and a 1-cm. vertical rupture with no evidence of healing in the left lateral esophagus about 2 cm. above the diaphragm. This was closed and underwater drainage of the chest instituted. On the seventh postoperative day the sutures were removed, and methylene blue was given by mouth; this appeared in the posterior tube, demonstrating a leak in the esophageal closure. Oral intake was stopped and intravenous therapy reinstituted. By the 12th postoperative day he was still draining profusely. The tubes in the midesophagus and stomach were replaced and connected to suction. He became afebrile on the 22nd postoperative day and the drainage slowly ceased. Nine weeks postoperatively fluids were given by mouth around the indwelling Levin tubes. A barium swallow two months postoperatively showed a small pocket of contrast media outside the esophagus on x-ray, although he was eating well and his general condition was excellent. The thoracotomy tubes were removed about two months postoperatively. He gained 15 lb. in weight and was discharged 22 weeks after admission.

Followup examination one year later showed a weight gain of 56 lb., no symptoms at full work and a normal chest x-ray and esophagogram.

Case 4 (Veterans Administration Hospital, Memphis, Tennessee)

History. This 62-year-old white male was admitted on April 28, 1956, with epigastric pain of several hours' duration *followed by* vomiting of about 500 cc. of blood. The patient was relatively asymptomatic at the time of admission. His past history dated back to 1952 at which time he was admitted for a cerebrovascular accident, left nephrolithiasis, left ureterolithiasis and chronic pyelonephritis. At that time a left nephroureterectomy was performed and he was readmitted in 1953 for fracture of the right femur. In January 1955 he was admitted for a chronic duodenal ulcer with hemorrhage. This was proved by x-ray, as was diverticulitis of the colon and pulmonary emphysema. Since May 1955, he had had intermittent vague indigestion, usually following meals and relieved by alkalis.

Physical examination. Physical examination was essentially within normal limits except for the following: blood pressure was 160/100; pulse 130; respirations 20; the lung fields were described as being clear; there was slight epigastric tenderness.

Laboratory data. Laboratory data on admission, including blood counts and blood chemistries, were normal. Portable chest film was interpreted as showing a mottling throughout both lung fields with a consolidation at the right base and a veiling at the left base. A scout film of the abdomen was negative. Electrocardiogram revealed a sinus tachycardia.

Course. The initial impression was bleeding chronic duodenal ulcer. The patient was placed on a medical regimen, but about 4 hr. later shock appeared and the patient became extremely confused. He was given 1,000 cc. of whole blood and norepinephrine was given. Following the transfusion, the hematocrit rose from 47 to 53, but shock continued. A Levin tube was passed into the stomach and a very slight amount of coffee-ground material was aspirated. Physical examination revealed a few rales in the left base and x-ray revealed an infiltration in the left base and some air on the left border of the heart shadow. It was felt that the patient had either a myocardial infarction or pulmonary infarction. He failed progressively in spite of vasopressors and died in cardiovascular collapse 15 hr. following admission.

Autopsy. The pertinent autopsy findings were as follows: The left pleural cavity contained 3,000 cc. of dark, muddy, reddish-brown fluid smelling of vomitus and the right pleural cavity contained 2,000 cc. of the same fluid. There were 1,500 cc. of muddy, reddish-brown fluid in the peritoneal cavity. The lungs were atelectatic. The left kidney was absent. At the gastroesophageal junction there was a 2-cm. perforation of the esophagus with dissection into the mediastinum by blood and gastric contents and rupture into both pleural cavities. The dissection extended down over the upper half of the stomach, forming a subserosal

pocket containing grayish-brown, muddy fluid. There were small areas of ulceration around the large perforation. The first portion of the duodenum showed an old healed ulcer.

Case 5 (Veterans Administration Hospital, Memphis, Tennessee)—previously reported[15]

History. A 55-year-old farmer was admitted on December 5, 1950, with a 7-hr. history of severe, sharp pain in the lower posterior chest bilaterally. This had occurred suddenly during an episode of coughing and was followed by a small amount of vomiting. The pain was aggravated by breathing and movement. There was weakness and the pain was not alleviated by morphine. He was known to have an obscure hemorrhagic disease resembling familial purpura simplex.

Physical examination. The patient was acutely ill, pale, cyanotic, and dyspneic, with a temperature of 97°F.; pulse 136; blood pressure 120/70 and respiration of 44. The right external jugular vein was very prominent. Percussion note was dull and breath sounds were diminished with rhonchi and rales over both lung bases. The abdominal muscles were spastic with generalized abdominal tenderness. Peristalsis was not heard. Rectal examination was noncontributory.

Laboratory data. Routine blood counts and urinalysis were normal. A chest x-ray showed pneumonitis in the left lower lung field.

Course. A Levin tube and urethral catheter were inserted. About 2 hr. after admission cervical subcutaneous emphysema was noted. An x-ray taken 5 hr. after admission revealed a left pleural effusion and blunting of the right costophrenic angle. A left thoracentesis showed dark brown, turbid fluid. Thoracotomy was not done because of the hemorrhagic disorder. A tube was inserted into the left pleural space and connected to underwater drainage and about 500 cc. of dark brown, turbid fluid was removed. Oxygen, penicillin, streptomycin and appropriate intravenous fluids were given. The rectal temperature rose to 104°F. and did not fall for several days. The blood pressure dropped to 50/0 with a pulse of 100, but rose to 70/50 the second day and to 120/80 the third day. The drainage subsided and the gastric suction was discontinued on the fourth day and the intercostal tube was removed on the fifth day. On the ninth day an x-ray following a swallow of iodized oil revealed air, fluid and oil in the posterior mediastinum. A segment of the eighth rib was resected and the empyema drained. Tube feedings were started, but some of the feedings appeared in the drainage. The heart shadow showed steady enlargement and diminished pulsations. Pericardial tap yielded 200 cc. of bloody fluid. Cardiac tamponade recurred and operation became mandatory. A large segment of the 1-cm.-thick pericardium was excised after evacuation of the bloody fluid and a catheter jejunostomy was done by a second team. The empyema was drained through a resected 11th rib. Jejunostomy feedings were begun on the second postoperative day and the right intercostal tube removed the following day. The temperature was normal the next day. The patient progressively improved. On the 29th postoperative day convalescent peptic ulcer diet was started. The jejunostomy tube was removed on the 32nd postoperative day. An incidental midesophagus diverticulum was found on x-ray and the deformity in the lower esophagus resulting from the perforation was seen.

Follow-up examination on November 1, 1956, showed no symptoms or significant abnormality of the esophagus.

*Case 6 (University Hospital, Birmingham, Alabama)**

History. This 55-year-old white male was admitted for elective cholecystectomy. Upper gastrointestinal symptoms had persisted for 10 years, characterized by vomiting and epigastric pain. In November 1954 a diagnosis of hiatal hernia was made and in December 1954 a repair of an esophageal hiatal hernia through a thoracoabdominal approach was done. At this time the gallbladder was inspected and the patient was advised to have a cholecystectomy after an interval of six weeks. Since the last admission there had been jaundice, and light stools and dark urine which disappeared in a few days without treatment.

Physical examination. Physical examination was essentially within normal limits except for a recent, well healed surgical scar on the left side, and minimal epigastric tenderness and pulmonary emphysema.

Course. On February 14, 1955, the patient underwent a cholecystectomy with exploration of the common duct. The duct was found to be normal. Four days postoperatively the patient began complaining of pain in the right upper quadrant which was aggravated by breathing. The blood pressure was elevated and he appeared apprehensive. Physical examination of the chest was described as normal. Chest x-ray showed no evidence of pulmonary infarction, but did reveal mediastinal emphysema which was interpreted as being the result of esophageal perforation. Some fluid was present in the right pleural cavity, and this was aspirated. A tube was inserted into the pleural cavity. Seven days postoperatively, crepitation appeared over the entire anterior chest, a friction rub was heard over the precordium, the pulse was 120 and he was icteric. The T-tube in the common duct was draining adequately. The patient passed

* Courtesy of Dr. Joe Butler.

a large copious stool which consisted of dark, old blood. On the eighth postoperative day the patient had a gastrostomy and drainage of the mediastinum. After the mediastinum was drained, the clinical course was progressively downhill, with cyanosis and peripheral vascular collapse which were poorly controlled with norepinephrine infusions. The temperature ranged from 103 to 105°F. rectally, in spite of large doses of antibiotics and what appeared to be adequate drainage. The blood counts and electrolytes were kept within normal limits by appropriate intravenous fluids and transfusions. He died on the tenth postoperative day.

Autopsy. Twelve hundred cc. of fluid was found in the right pleural cavity. The upper two thirds of the esophagus was normal, but the lower third showed several fresh small shallow mucosal ulcerations. At the esophagogastric junction there was a 2-cm., sharp, recent perforation which communicated with the right thoracic cavity. The area of mediastinal inflammation appeared to connect inferiorly with massive retroperitoneal necrosis. The liver appeared grossly normal. The T-tube was draining and the common duct appeared unobstructed. The autopsy diagnosis: (1) spontaneous perforation of the esophagus with empyema and mediastinitis; (2) homologous serum jaundice.

Comment. This patient had a previous hiatal hernia and this may conceivably have terminated in the perforation. However, the lower esophagus was seen and felt only two months before the perforation and no abnormality was noted.

Case 7 (Veterans Administration Hospital, Memphis, Tennessee)

History. This 60-year-old white male alcoholic drank heavily, almost daily, until November 11, 1956, at which time he vomited a large amount of bright red blood. This was followed by tarry stools for several days, and upon a further hemoptysis three days later, he was admitted to another hospital where he was treated medically with multiple blood transfusions for a bleeding peptic ulcer. On December 5, 1956 crepitation was found in his neck and a diagnosis of ruptured esophagus was made. A left exploratory thoracotomy was done and a rupture in the lower esophagus sutured. Two large tubes were placed in the mediastinal and pleural spaces. High fever persisted and purulent drainage soon escaped from his thoracotomy tubes. He was transferred to Kennedy on his seventh postoperative day.

Physical examination. Complete physical examination was essentially normal except for the following: Temperature of 100°F., pulse 100 and blood pressure 160/90. The patient was an emaciated, acutely ill white male who was somewhat confused mentally. The palms and plantar surface of the feet were beefy red. The skin was hot and dry and the tongue longitudinally furrowed. There were two large thoracotomy tubes issuing from the left midaxillary line. The operative wound in the left chest was well healed. A few rales were heard in the left base posteriorly. The abdomen appeared slightly distended and the liver was felt about 1 cm. below the right costal margin and was tender.

Laboratory data. The white blood count was 19,100 with a left shift. Urinalysis showed a trace of albumin with 10–12 white blood cells and 50–60 red blood cells per high powered field. Total protein was 5.1 gm. with 2.2 gm. of albumin. Blood chemistries were within normal limits except for a 2.8 serum potassium. X-ray of the chest showed marked widening of the mediastinum with free air and diminished aeration of the left lower lung field.

Course. Penicillin, streptomycin and intramuscular chloromycetin were administered. Thick purulent material continued to drain from the thoracotomy tube. A double-lumen tube was placed in the duodenum and stomach allowing gastric suction and feeding into the distal duodenum, but this proved unsatisfactory because of regurgitation of the feeding into the stomach. On December 21 and 23, 100 mg. of intravenous hydrocortisone were given. The electrolyte imbalance was corrected by intravenous fluids. On December 21 the patient developed jerky movements of both arms and hands and became irrational. This persisted until December 23, when he became cyanotic and had a marked fall in blood pressure. Large amounts of dark blood were aspirated from the stomach tube. Blood and Levophed were administered, but the blood pressure could not be maintained. His respirations became labored and shock persisted until he died on December 24. It was believed that the patient's death was caused by gastrointestinal hemorrhage from gastrointestinal ulcerations.

Autopsy findings. The suture line in the esophagus was well healed and intact. There was a widespread suppurative mediastinitis, lobular pneumonia and septicemia. There were multiple ulcerations in the stomach, duodenum, small bowel and colon. There was a 3-cm. penetrating ulcer in the lesser curvature of the stomach. The remainder of the small bowel and colon showed linear ulcerations with necrotic bases extending well into the muscularis, some of these being as much as 25 cm. in length.

A tabulation of some significant aspects of these cases is presented in table I.

TABLE I

Case	Age	Onset	Vomiting	Dyspnea at Onset	Previous Gastrointestinal Disease	Duration before Hospitalization	Admitting Diagnosis	Shock	Abdominal Tenderness	Emphysema	Outcome
	yr.					*hr.*					
1	40	During coughing; sudden severe epigastric pain	Gross hematemesis *following* pain	2+	Duodenal ulcer	4	Perforated duodenal ulcer	On admission	Epigastric; severe	Cervical; 6 days after onset	Died 13th day
2	53	Sudden anterior chest pain while standing and talking	None	4+	None	2	Myocardial infarction	9 hr. after onset	None	None	Died after 11 hr.
3	46	Slow, *after* vomiting; dull epigastric and bilateral lower chest pain	First symptom; *no* Blood	3+	None	96	Infected pulmonary cysts; empyema	None	Left upper quadrant, mild	Mediastinal; 5 days after onset	Cured
4	62	Slow epigastric pain	Gross hemorrhage *after* pain and dyspnea	1+	Duodenal ulcer; diverticulosis of colon	4	Bleeding duodenal ulcer	8 hr. after onset	Epigastric; mild	None	Died after 19 hr.
5	55	Sudden onset during coughing; sharp lower, posterior thoracic pain	*After* pain	4+	None	7	Pneumonia	10 hr. after onset	General; severe	Cervical; 9 hr. after onset	Cured
6	55	4 days postoperatively; cholecystectomy; slow onset; right upper quadrant pleuritic pain	Yes—association with onset questionable	1+	Hiatal hernia (postoperative); cholecystitis (postoperative)	120	Postoperative	None	? Postoperative	Mediastinal; 48 hr. after onset	Died 6th day
7	60	Gross hematemesis without other symptoms	First symptom	1+	Gastric ulcer	72	Bleeding duodenal ulcer	None	Epigastric; mild	None	Died 23rd day

SUMMARY

Seven patients with spontaneous rupture of the esophagus, not having the benefit of early operation, are reported. The history and physical examination were atypical, with pain present in only 2 patients, and vomiting related to onset in only 2 patients. The diagnosis was initially in error in all of these patients, 5 of whom died.

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INTUSSUSCEPTION: A REPORT OF 148 CASES*

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Intussusception is the invagination, slipping, or passage of one part of the intestine into another. The intussusceptum is the invaginated portion of the intestine, and the intussuscipiens is that portion of the intestine receiving the other segment. The ileocecal intussusception is one which takes place at the ileocecal valve with the cecum, as well as the ileum, being invaginated into the colon. Enteric intussusception is an intussusception involving the small bowel only. Colic intussusception is an intussusception involving the large bowel only. The incidence with which intussusception occurs varies widely in different countries. In England, Denmark, and Australia it constitutes one of the most frequent varieties of intestinal obstruction. In the United States it appears to be definitely less frequent. However, in a series of 110 cases of intestinal obstruction reported by Wilson, Hardy and Farringer, intussusception ranked third in frequency.[5] About 75 per cent of all patients with the disease are children under two years of age. In this series of 148 patients with intussusception, 70 per cent were in the age group of 4 to 24 months. In infants an etiologic agent is rarely demonstrated. In the adults, however, an etiologic agent is found in approximately 70 per cent. Polyps and Meckel's diverticula were the lead points in 8 of 11 adults in this series. Intussusception occurs most frequently at the ileocecal junction when vigorous peristaltic waves are countered by antiperistaltic waves in the lower ileum. Antiperistaltic waves normally occur in the duodenum and lower ileum. Intussusception is almost invariably of the descending variety, but ascending varieties do occur. There were no patients with the latter type in this study.

The obstruction in intussusception is afforded by the active contraction of the sheath, especially at the neck. The usual intussusception consists of three cylinders. The internal and returning layers constitute the intussusceptum. The outer tube is known as the intussuscipiens or ensheathing layer.

The greatest alteration in the bowel in acute intussusception occurs in the intussusceptum. The walls of the invaginated segment rapidly become edematous and engorged with blood as a result of venous obstruction in the involved mesentery. Later, ulceration, gangrene and perforation may develop because of arterial insufficiency. Circulatory embarrassment is the factor which accounts for the "currant jelly" type of blood in the stool after the first 10 to 12 hr.

There are four cardinal points in the diagnosis of intussusception: periodic attacks of pain, vomiting, passage of mucus and blood by rectum, and the presence of a palpable tumor. In a series of 41 cases of acute intussusception reported by Santulli and Ferrer, an abdominal mass was palpated in 90 per cent of the patients.[3] The usual history is that an apparently healthy infant is suddenly seized with agonizing abdominal pain. The abdominal pain persists for a short time and then disappears. It is relentless, however, and returns in episodes separated by periods of quiescence and comparative ease. Frequently the child will sleep between the episodes of pain. Vomiting occurs with the onset but is usually not persistent. The excited bowel may pass normal feces during the first few hours, but after a variable period of time, often 10 or more hours, there may appear the characteristic "current jelly" type blood with mucus in the stool. The general appearance of the patient changes as the condition progresses. The infant who is distressed and hyperactive in the early stages is gradually transformed to a state of apathy and prostration. At first, the patient is afebrile, but after 24 hr. have elapsed fever develops as a result of dehydration and possibly infection.

The mass formed by the intussusception is curved, sausage shaped, and located along the course of the colon. The location of the mass depends upon the site of origin, the speed with which it progresses, and the time from onset to

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examination. It will occasionally progress down to the rectum. On digital examination one can feel the intussusceptum which gives the sensation of the uterine cervix. In one case of intussusception, reported by Dunavant and Wilson, the appendix presented at the anus.[1]

If the diagnosis is equivocal a barium enema should be given and x-ray studies made. This examination will demonstrate complete obstruction of the colon, and may show the typical coiled-spring effect caused by barium in the crevices between the invaginated and receiving portions of the intussusception.

In 1905, Hirschsprung gained world wide renown for his successful reduction of intussusception by administration of barium enema. The barium enema was given with a 3-foot gravity pressure.[4] Hughes listed the following points against the use of this method of reduction of an intussusception: (1) if the bowel has already become gangrenous it may be ruptured by the pressure of the enema, (2) the time used for this procedure may allow viable bowel to become gangrenous, (3) barium enema cannot reduce intussusception limited to the small bowel, (4) barium enema will usually not reveal the occasional predisposing factor such as a polyp or a Meckel's diverticulum.[2] The disadvantages listed by Santulli and Ferrer are the same as the above.[1]

The treatment of choice is surgical reduction of the intussusception. Fluids and electrolytes can usually be restored satisfactorily while arrangements are being made for the operation. In a late case where peritonitis, shock, and dehydration are present, a delay may be necessary for restoration of more adequate fluid and electrolyte balances. The mortality rate is low for those patients on whom surgery is done in the early hours of the disease. If operation is delayed for more than a day the mortality rate is very high.

After correction of dehydration, the abdomen is prepared with ether and tincture of zephiran. If the operation is performed in an infant over 24 hr. of age, the necrotic remnant of the cord is a definite hazard, and should be excluded from the field of surgery to prevent peritoneal contamination. A midline or paramedian abdominal incision is made under ether anesthesia. The cause of the intestinal obstruction is readily found

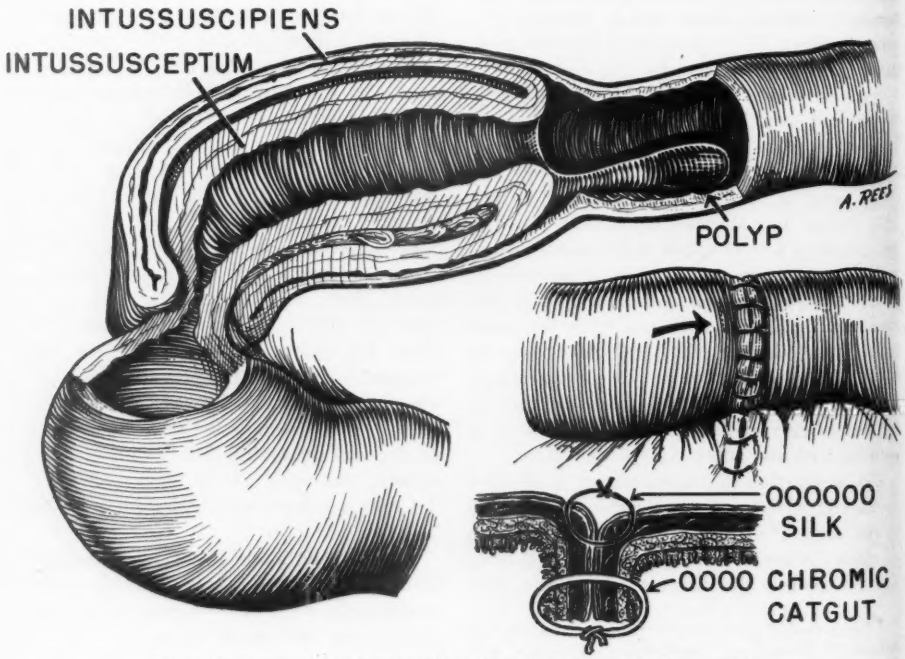

FIG. 1. Diagrammatic drawing of method of anastomosis after resection

on exploration of the abdominal cavity, and appropriate steps are taken to correct the abnormality. Other abdominal pathology should not be overlooked. In some instances of intussusception, simple reduction alone is necessary. In those patients with irreducible intussusception, resection and anastomosis must be done. In the reconstitution of the continuity of the intestine after resection, a single layer of silk suture is used when the diameter of the distal segment of intestine is small. This occurs most often in those patients with atresia or stenosis. In most cases, however, two layers of sutures should be used. In the infant, we recommend an inside layer of through-and-through individual sutures of No. 0000 chromic catgut placed near the severed margins of the intestine and 4 to 5 mm. apart. The outside layer is made with No. 00000 or 000000 arterial silk on an atramatic needle. The sutures are placed 3 mm. apart, and care is taken not to pass the needle through the full thickness of the bowel wall. The sutures should be tied only tight enough to just approximate the serosal surfaces. One must remember that edema always occurs at the site of anastomosis after resection of a segment of intestine. If the anastomosis is made carelessly, there may be leakage and subsequent peritonitis. If too much margin is rolled in, a partial diaphragm is easily produced which may result in further obstruction. Because the diameter of the intestine of the infant is so small the latter is a definite possibility. Gastric suction is maintained for a few days following surgery. Parenteral administration of fluid is continued until the patient is able to take fluid by mouth. Antibiotics are given before surgery and for four to five days after surgery (fig. 1).

CASES

The case reports in this study were taken from the medical records of Vanderbilt, St. Thomas,

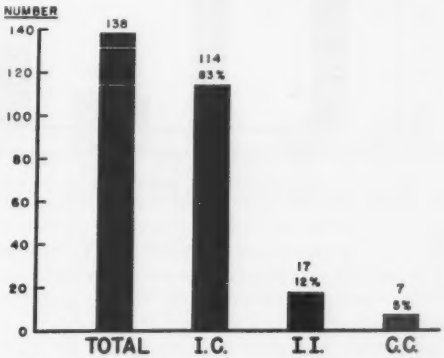

FIG. 2. Graph shows the types of intussusception in this series of 138 patients. The greatest number is in the ileocolic classification.

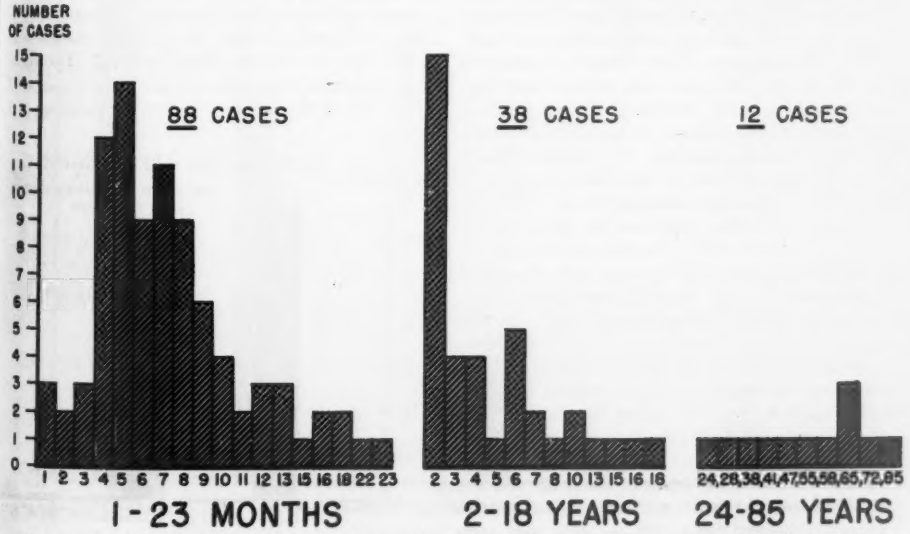

FIG. 3. Graph shows age incidence in this series of 138 patients with intussusception. Note the large number in the first two years of life.

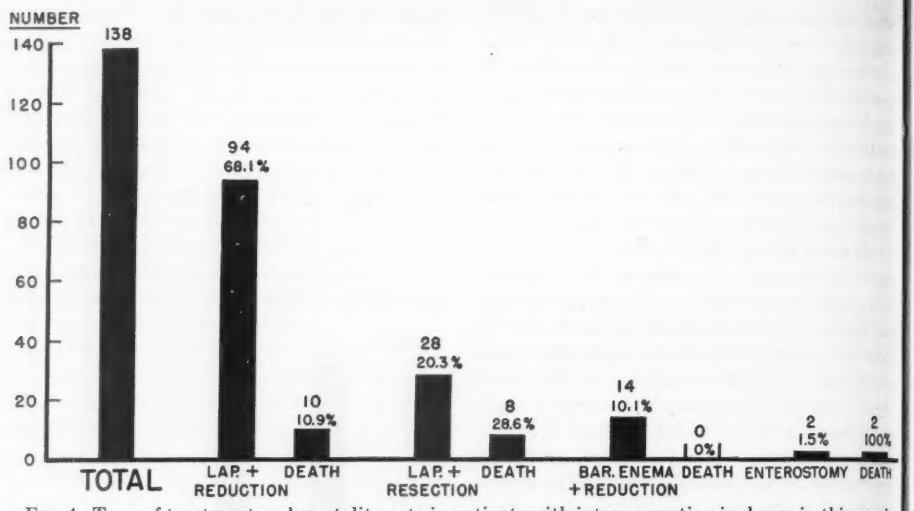

FIG. 4. Type of treatment and mortality rate in patients with intussusception is shown in this graph. Note the high mortality when resection was necessary.

and Baptist Hospitals in Nashville, Tennessee. The records at Vanderbilt cover the period from 1927 to 1957. The records at St. Thomas and Baptist Hospitals cover the period from 1946 to 1957. A summary of the entire series will be given followed by a comparison of those patients with intussusception at Vanderbilt from 1927 to 1946 with those patients with the same disease from all three hospitals from 1946 to 1957.

The series consists of 148 patients with intussusception. In 10 patients the diagnosis was made by clinical examination only. These are excluded from the study. The other cases were proved diagnoses made by x-ray studies or by exploratory laparotomy. Eighty-nine or 64 per cent of 138 patients with intussusception were males. Forty-nine or 36 per cent were in females. This gives a sex incidence of males to females of approximately two to one. A further study of the sex incidence indicates that the ratio for males over females up to the age of 10 years is two to one. After the age of 10 years, however, the sex incidence is one to one. Interesting speculation can be made as to why this is so. Is it that sex hormones play an important part in the production of intussusception? We believe that a far greater series of patients with this disease must be studied before this fact can be fully determined. Most writers give a ratio of two to one for males under one year of age. A few give a ratio of three to one in favor of the male.

The percentage mortality for the entire series was 17.6 per cent, with 17.9 per cent mortality for males and 17 per cent mortality for females. In 138 patients the type of the intussusception was recorded. The predominant type was the ileocolic which was present in 114 patients or 83 per cent. The next most common type was the enteric which was present in 17 patients or 12 per cent. The least common type was the colic of which there were only 7 patients, or 5 per cent (fig. 2). The age range was from 5 days to 85 years. There were 95 patients or 69 per cent in the age range of 4 through 24 months (fig. 3).

In 14 patients or 10.1 per cent the intussusception was reduced when the barium enema was given. All of these patients survived. The most common means of treatment was laparotomy with reduction of the intussusception or resection of a

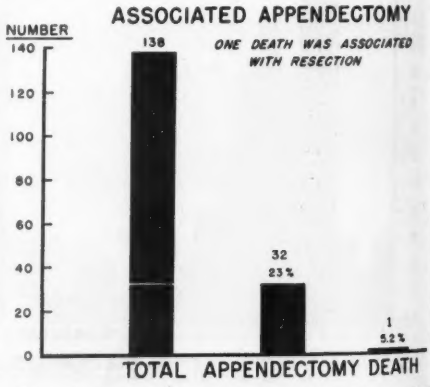

FIG. 5. Appendectomy apparently did not increase the mortality rate in this series as indicated in this graph.

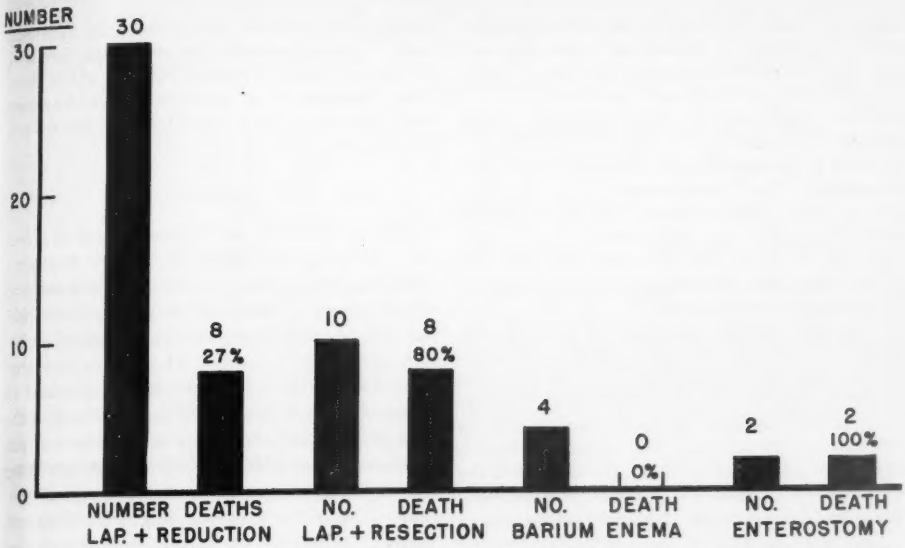

FIG. 6. The treatment of intussusception is indicated in this graph of 46 patients from Vanderbilt Hospital from 1927 to 1946. Note the high mortality rate of 39 per cent.

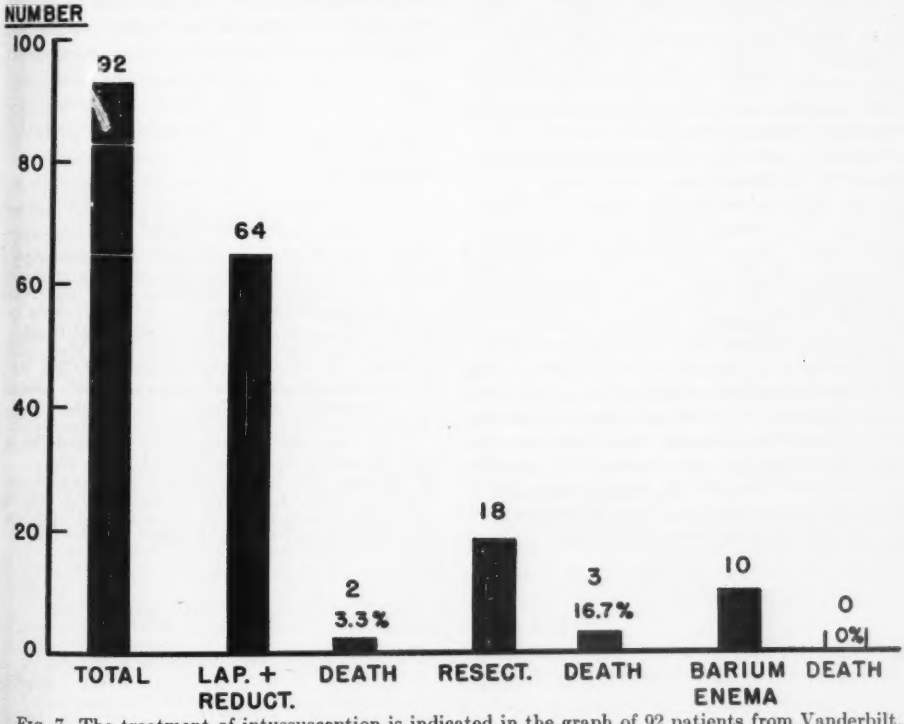

FIG. 7. The treatment of intussusception is indicated in the graph of 92 patients from Vanderbilt, St. Thomas and Baptist Hospitals from 1946 to 1957. Note the lower mortality rate of 5.4 per cent.

segment of intestine. Ninety-four or 68.1 per cent of the patients were treated by laparotomy and reduction. Ten of these patients died—a mortality rate of 10.7 per cent. In 28 patients it was necessary to resect a segment of the intestine. Eight of these patients died—a mortality rate of 28.6 per cent. There is an increase in the mortality with delay in operation. Two patients were treated by enterostomy alone. Both of these died. Thirty-two of the patients had an associated appendectomy. Only one of these died following resection. We believe that appendectomy adds no additional risk to the operation (figs. 4 and 5).

From 1927 to 1946, there were 46 patients with intussusception treated at Vanderbilt Hospital. Twenty-nine of these patients or 63 per cent were males and 17 or 37 per cent were females. Twenty-eight of the patients survived, and 18 died—a mortality of 39 per cent. The types of intussusception in this group are much the same as in the entire series. Two of the patients had colocolic, three had ileoileal, and 42 had ileocolic types of intussusception. Four of these patients or 8.6 per cent were treated by closed reduction by means of barium enema. There were no deaths in these 4 patients. Thirty patients were treated by laparotomy and reduction of the intussusception. Eight of these died—a mortality of 27 per cent. In 10 patients explored, resection was necessary. Eight of these died—a mortality of 80 per cent. The 2 patients treated by enterostomy died, giving a mortality rate of 100 per cent (fig. 6).

From the years 1946 to 1957, there were 92 patients with intussusception treated in the Vanderbilt, St. Thomas, and Baptist Hospitals. Sixty or 65.2 per cent were males and 32 or 34.8 per cent were females. Eighty-seven patients survived and 5 died—a total mortality of 5.4 per cent. This is a favorable improvement over the 39 per cent mortality in 46 patients treated at Vanderbilt for the previous 19 years. Sixty-three of these patients were between the ages of 4 and 24 months. The types of intussusception during this period are in close agreement with the other groups. Seventy-nine per cent were ileocolic, 15 per cent were ileoileal, and 5 per cent were colocolic in type. Ten patients were treated by closed reduction by means of barium enema. These 10 patients survived. Sixty-four patients were treated by laparotomy and reduction. Two of these or 3.1 per cent died. Eighteen patients required resection of a segment of bowel. Three or 16.7 per cent of these died. There were 23 associated appendectomies with laparotomy and reduction, and all of these patients survived (fig. 7).

SUMMARY

The definition of an intussusception is given. The pathology, the diagnosis, and the treatment are discussed. A study of 138 cases is presented. From this study which covers a period from 1927 to 1957 we note a marked improvement in the mortality rate in the last 11 years as compared with the previous 19 years. In the previous 19 years the mortality was 39 per cent and in the last 11 years only 5.4 per cent. The cause of this decrease in mortality can probably be attributed to: (1) early diagnosis, (2) better surgical techniques, (3) better understanding of fluid balances, and (4) antibiotics. It is interesting to note that the sex incidence up to the age of 10 years is two to one in favor of the male after which the sex incidence decreases to a ratio of one to one. If this is a true observation, further studies are indicated to determine the cause. Associated appendectomy produces no increase in mortality rate.

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A CRITICAL EVALUATION OF POLYVINYL SPONGE (IVALON) AS A VASCULAR AND TISSUE SUBSTITUTE*

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INTRODUCTION

With the continued impetus in the search for the "ideal" vascular and tissue substitute, polyvinyl sponge (Ivalon) has come under our scrutiny.

The first reported clinical use of Ivalon was in 1949 by Grindlay and Clagett when they applied the sponge to fill intrathoracic defects following pneumonectomy.[6] In 1955, Shumway, Lewis and Gliedman[14] demonstrated a further use of Ivalon in the construction of arterial grafts from compressed tubes of the sponge. Lewis and Taufic[10] in 1955 used tailored sheets of the polyvinyl sponge for the repair of interventricular septal defects.

Our first report on the use of Ivalon as an arterial graft emphasized the methods of arterial graft construction, and stressed the superb physical and technical properties of this plastic (Jesseph, Stevenson and Harkins, 1956).[7] Since that time we have utilized Ivalon in a number of ways, including vascular grafts in the thoracic aorta, autocompilation aortic grafts in growing pigs, pulmonary artery grafts, long aortic bypass grafts, long medium sized arterial bypass grafts, superior vena cava grafts, tracheal and esophageal prosthesis, repair of tissue defects (diaphragm) and as a hemostatic agent in the repair of liver and kidney wounds. On the basis of reports of Ivalon failure in a number of these areas, we felt that a critical reevaluation of Ivalon as used in a number of specific instances was warranted.

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METHOD

A total of 113 mongrel dogs and 8 growing pigs was used in this entire study; 56 dogs and 8 pigs were included in the vascular study, 20 dogs in the liver and kidney hemostasis study, 14 in the tracheal prosthesis study, 8 in the diaphragmatic replacement study and 5 in the esophageal prosthesis study.

The Ivalon used for grafts and hemostatic patches and tissue defect replacement was prepared as described by Jesseph, and associates (1956)[7] and Benjamin and associates (1957).[2]

The animals were given the usual general anesthetic of phenobarbital. Mechanical respiration was utilized for those animals undergoing open chest procedures.

End-to-end grafts of the aorta were sutured in place with either a continuous over-and-over suture or a continuous everting mattress suture of No. 5-0 silk. The bypass grafts of the aorta were fashioned and sutured in place by the end-to-side method of Linton (1955).[11] The Ivalon compilation arterial grafts in the 8 growing pigs were fashioned after the method of Griffith and associates (1955).[5] The aorta was occluded for 10 to 20 min. while the various aortic anastomoses were made. Included in the aortic graft study were 19 dogs in which Ivalon bypass grafts were placed between the thoracic and abdominal aorta. In 8 of the animals, the aorta was ligated and transected just below the proximal (thoracic) anastomosis. In the remaining 11 animals, the aorta was ligated just above the distal (abdominal) suture line.

The use of Ivalon in patches as a method of hemostasis in liver and kidney wound repair was evaluated in 20 dogs. The methods used in this study are reported elsewhere (Jones and associates, 1957).[9]

The use of Ivalon as a tracheal prosthesis was reported from this laboratory in a previous preliminary report by Jesseph and co-workers (1957).[8] This present report includes a follow-up sum-

mary on these 14 animals. Ivalon was also utilized as an esophageal prosthesis in 5 animals. The prosthesis constructed was very similar in nature to the tracheal prosthesis as reported by Jesseph, the difference being the incorporation of a solid sheet of polyvinyl plastic sheeting between the layers of Ivalon.

The final group of 8 animals comprises an evaluation of Ivalon as a tissue defect replacement. In this study one leaf of the diaphragm was partially excised and a tailored sheet of mildly compressed Ivalon was sutured circumferentially into the defect.

TABLE I

Ivalon thoracic aortic grafts

Number of Dogs	Dimensions of Grafts (mm.)			Fate of Grafts
	Length	Diameter	Thickness	
11	20–30	13–14	1	3 dogs alive and well at present time; 3 dogs died within 2 to 20 days postop. from graft dilation and rupture; 5 dogs were sacrificed 3 months to 1½ years following operation; all grafts dilated to 25 to 50% of original diameter; all lined by endothelium

RESULTS

Thoracic aortic replacements (*tables I and II*). Of the 11 animals used in this study, 3 dogs are alive and well with patent grafts at the present time (2 years following operation) and 3 animals died within 2 to 20 days after operation from graft dilation and rupture (fig. 1). The remaining 5 dogs were sacrificed 3 months to 1½ years following operation. These grafts showed a 25 to 50 per cent dilation from their original diameter. All revealed Ivalon invasion by fibrous tissue and were lined by endothelium.

The 8 growing pigs with thoracic aorta compilation grafts of Ivalon were sacrificed 2 months to 6 months after operation. Only one graft showed any degree of dilation. The remaining grafts showed isodimensional growth in comparison to the host aorta, and were in excellent condition (fig. 2).

Small arterial replacements (*table III*). Eight dogs received pulmonary artery grafts. These animals were sacrificed from 1 week to 9 months postoperatively and all grafts were found to be thrombosed (fig. 3).

Aortic bypass grafts (*table IV and V*). Nineteen dogs were given thoracic abdominal aortic bypass grafts 190 to 350 mm. in length. In 8 of the

TABLE II

*Ivalon auto compilation thoracic aortic grafts**

Pig No.†	Graft Dimensions at Operation				Length of Follow-up	Graft Dimensions at Time of Death or Sacrifice				Fate of Grafts
	Length	U.A.	M.A.	L.A.		Length	U.A.	M.A.	L.A.	
	mm.				*mo.*	*mm.*				
384–1	20	11	11	11	2	26	14	14	13	Graft in excellent condition ± dilation
385–1	19	12	12	11	6	21	17	16	17	Graft in excellent condition ± dilation
386–1	22	12	13	12	6					
387–1	25	13	12	12	6	24	19	20	18	Graft in excellent condition ± dilation
388–1	24	12	12	12	6					
389–1	22	13	13	12	6	22	19	19	18	Graft in excellent condition ± dilation
390–1	24	14	14	14	2	26	16	17	16	Graft in excellent condition ± dilation
391–1	22	13	13	13	2	23	16	18	16	Slight dilation of Ivalon strip

* Ivalon compilation strip: 1 mm. thick, 8–10 mm. wide, 19–25 mm. long.
† Total number of animals (pigs) = 8.

FIG. 1. Ivalon thoracic aortic grafts showing the rather marked dilatation and rupture which occurred in 3 animals.

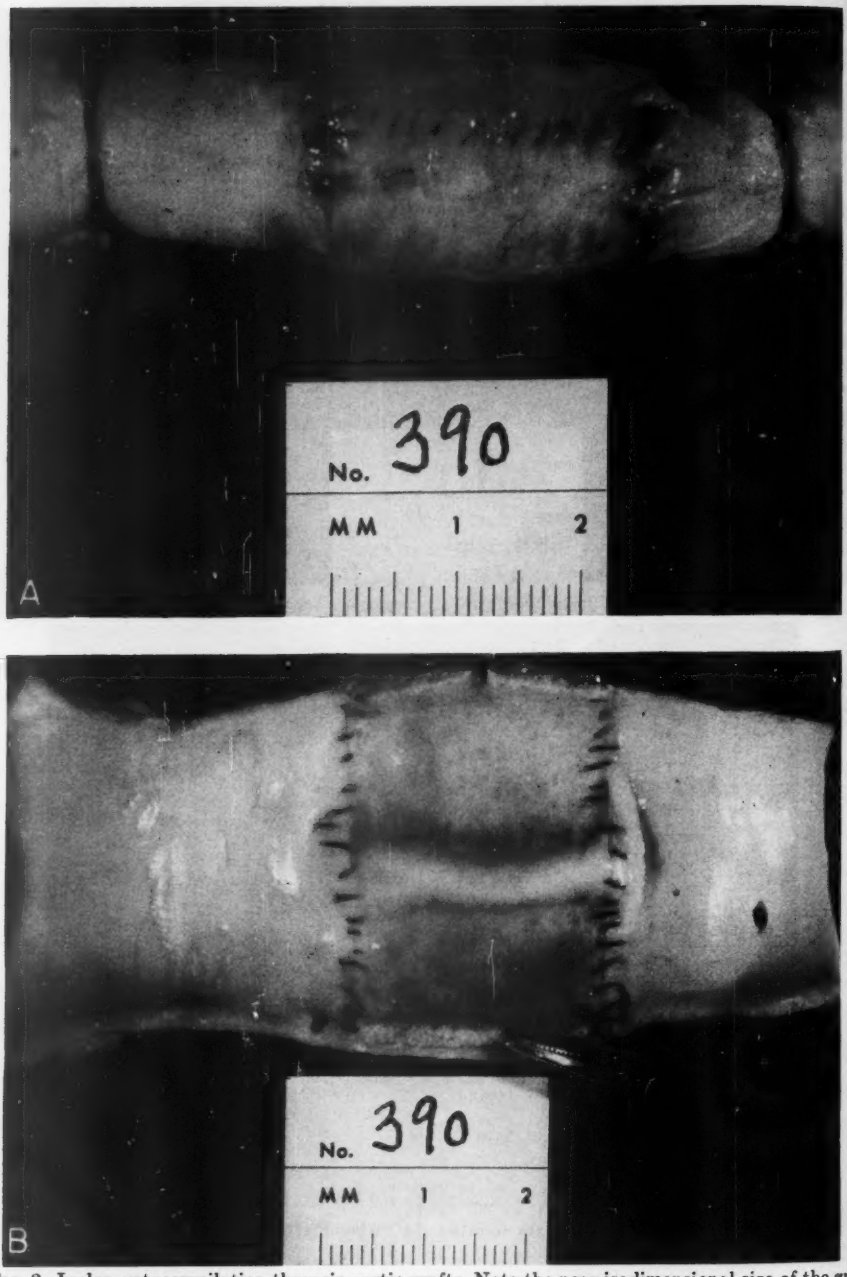

FIG. 2. Ivalon autocompilation thoracic aortic grafts. Note the near isodimensional size of the graft segment with that of the adjoining host aorta.

TABLE III

Ivalon pulmonary artery grafts

Number of Dogs	Dimensions of Grafts (mm.)			Fate of Grafts
	Length	Diameter	Thickness	
8	8–30	5–7	1	All of the grafts thrombosed; animals that survived were observed for 1 week to 9 months following operation

TABLE IV

*Ivalon aortic bypass grafts** (Thoracic-abdominal aorta)

Number of Dogs	Dimensions of Grafts (mm.)			Fate of Grafts
	Length	Diameter	Thickness	
2	190–270	7	1–1.5	Grafts open and functioning at the end of 6 weeks' follow-up; all lined by endothelium
6	275–350	7	1–1.5	Grafts thrombosed. Animals survived and were observed for 2 days to 6 months following operation

* Aorta ligated below the proximal anastomosis.

TABLE V

*Ivalon aortic bypass grafts** (*thoracic-abdominal aorta*)

Number of Dogs	Dimensions of Grafts (mm.)			Fate of Grafts
	Length	Diameter	Thickness	
3	240–330	8–10	1–1.5	Grafts open and functioning at the end of 1 year's follow-up; all lined by endothelium
5	240–300	5–8	1–1.5	Grafts thrombosed; animals survived and were observed for 3 days to 1 year following operation

* Aorta ligated just above the distal anastomosis.

animals in which the aorta was ligated and transected just below the proximal anastomosis, the grafts were patent and functioning at the end of 6 months' follow-up. The remaining 6 grafts were observed for 2 days to 6 months following operation and all were thrombosed. In the remaining 11 animals, in which the aorta was ligated and transected just above the distal anastomosis, 3 grafts were open and functioning at the end of 1 year's follow-up (fig. 4). The remaining 8 grafts were observed for 3 days to 1 year after operation and these were also thrombosed (fig. 5).

Small arterial bypass grafts (*table VI*). There were 5 dogs in this series, 3 of which had bypass grafts between the left subclavian and left femoral artery. The remaining 2 dogs had grafts between the left subclavian and left common iliac artery. All of these 350- to 500-mm.-long bypass grafts thrombosed in the early postoperative period.

Large vein (*superior vena cava*) *grafts* (*table VII*). Included in this series of 13 dogs are 6 animals with Ivalon grafts, 3 with autogenous vein grafts and 4 with autogenous pericardial grafts. All of the grafts were occluded by a perivascular

TABLE VI

Ivalon long aortic bypass grafts

Number of Dogs	Type of Graft	Dimensions of Grafts (mm.)			Fate of Grafts
		Length	Diameter	Thickness	
3	Left subclavian artery to left femoral artery	410–500	5	1–1.5	All of the grafts thrombosed in the early postop. period
2	Left subclavian artery to left common iliac artery	350–465	5	1–1.5	All of the grafts thrombosed in the early postop. period

TABLE VII

*Ivalon superior vena cava grafts**

Number of Dogs	Dimensions of Grafts (mm.)			Fate of Grafts
	Length	Diameter	Thickness	
6	20–32	7–10	1	All of the grafts thrombosed in the early postop. period; animals survived and were observed for 2 weeks to 4 months following operation

* To be included in this group are 7 other animals with superior vena cava grafts (3 autogenous vein and 4 autogenous pericardium). The results were identical to those with Ivalon.

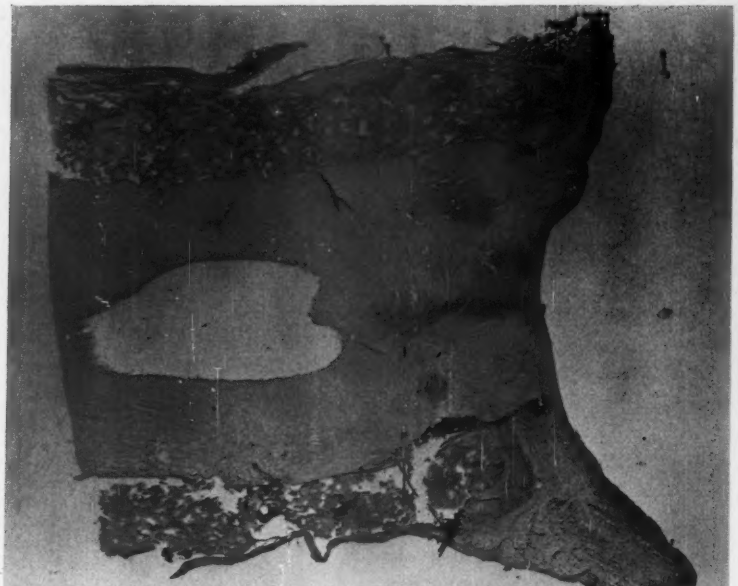

FIG. 3. Ivalon pulmonary artery graft. *A*. Longitudinal view of the gross specimen demonstrating a well organized thrombus. *B*. Microscopic section of the same specimen demonstrating the well organized thrombus as well as the healing of the anastomosis.

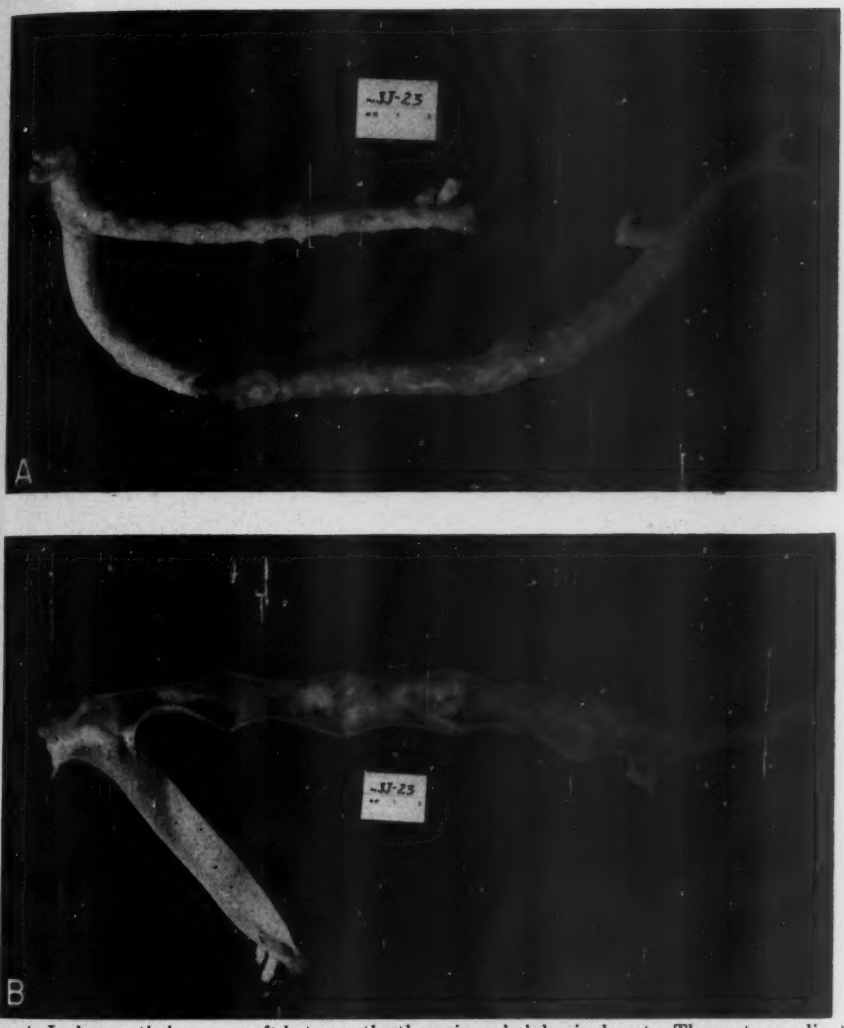

FIG. 4. Ivalon aortic bypass graft between the thoracic and abdominal aorta. The aorta was ligated and transected just above the distal anastomosis. Note in this case the excellent healing and over-all condition of the graft (graft diameter 10 mm.).

constriction of fibrous tissue at the area of grafting, 2 weeks to 4 months after operation.

Replacement of trachea and esophagus. Of the 14 animals with tracheal replacements, 2 are still alive and well at the present time (2 years after operation). The remaining 12 animals died at various intervals over the 2-year period following operation. The 5 animals with esophageal replacement all died within 2 months of operation. Failures in all of these animals were due to a variety of complications—leakage, graft sloughs, infection and scar stricture at the site of replacement (figs. 6 and 7).

Diaphragm replacement. Of the 8 animals in this series, all were sacrificed 1 year following operation. All of the Ivalon patches were intact and well healed, without evidence of hernia or infection. Whereas some other plastics tend to fragment in the diaphragm, there was no fragmentation of the Ivalon. There was a minimal

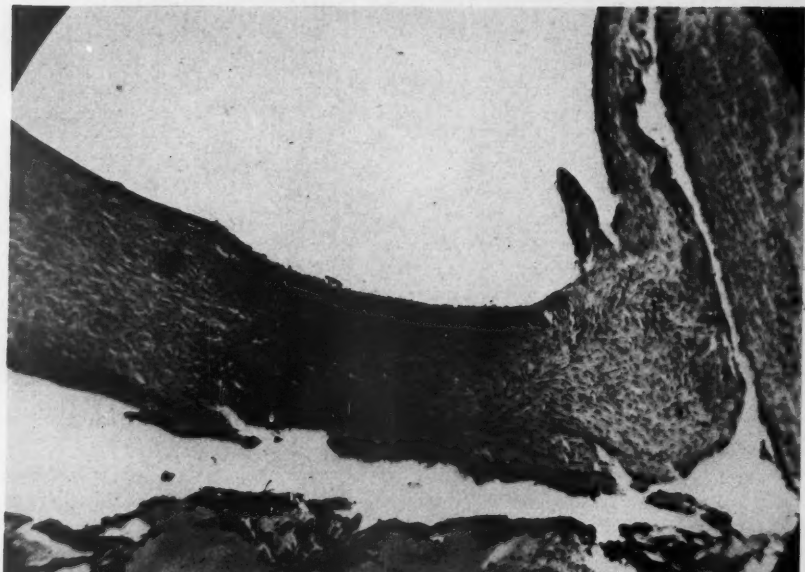

FIG. 5. Ivalon aortic bypass graft between the thoracic and abdominal aorta. The aorta was ligatee and transected just above the distal anastomosis. Note in this case the well organized thrombus in thd lower and upper end of the graft (graft diameter 7 mm.). *A*. Gross specimen opened by a longitudinal incision of the graft and host aorta. *B*. Microscopic section of the specimen taken at the area of the lower anastomosis. Notice the degree of healing and thickness of the endothelium (separation of the endothelium and Ivalon is an artifact due to the sectioning process).

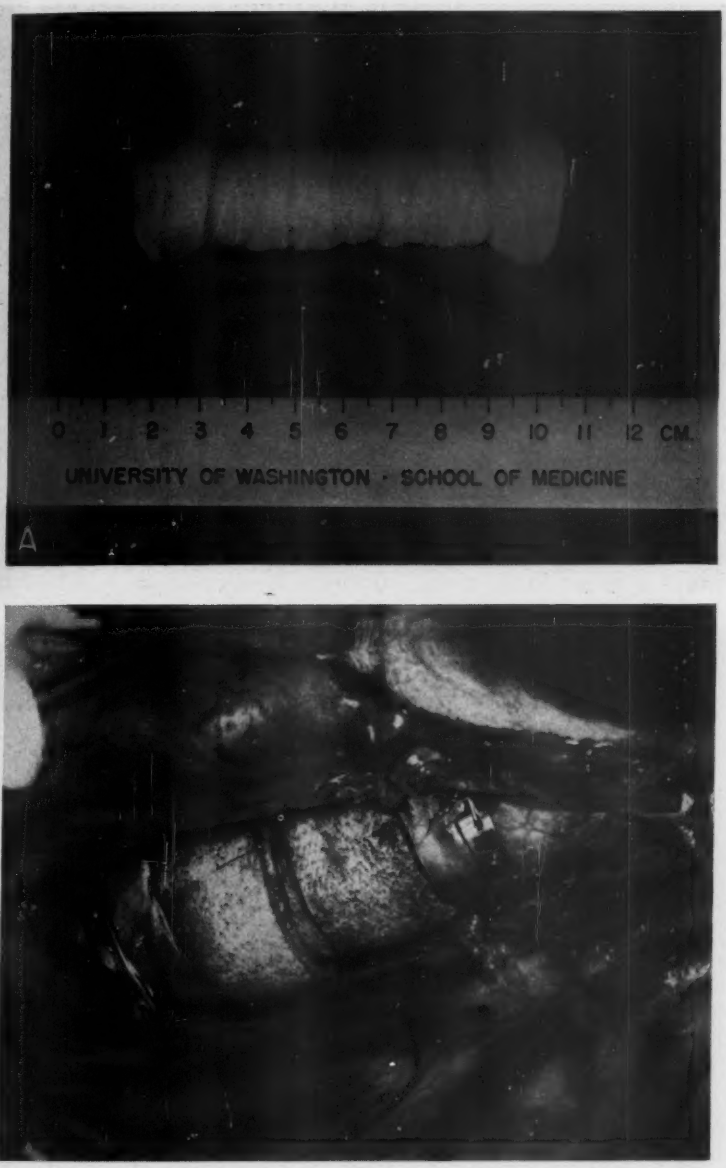

FIG. 6. Tracheal prosthesis. *A*. Completed prosthesis with cuffs turned down. *B*. Prosthesis in place with the cuffs turned back illustrating the proximal and distal ends of the trachea and the multiple pin fixation bands of stainless steel.

to a moderate number of adhesions between the Ivalon and the lung above and stomach below (fig. 8).

Liver and kidney wound repair with Ivalon. There were 20 animals in this series, with 2 deaths and 1 instance of infection, none of which were directly due to the Ivalon. Excluding the 2 deaths above, all of the animals were alive and

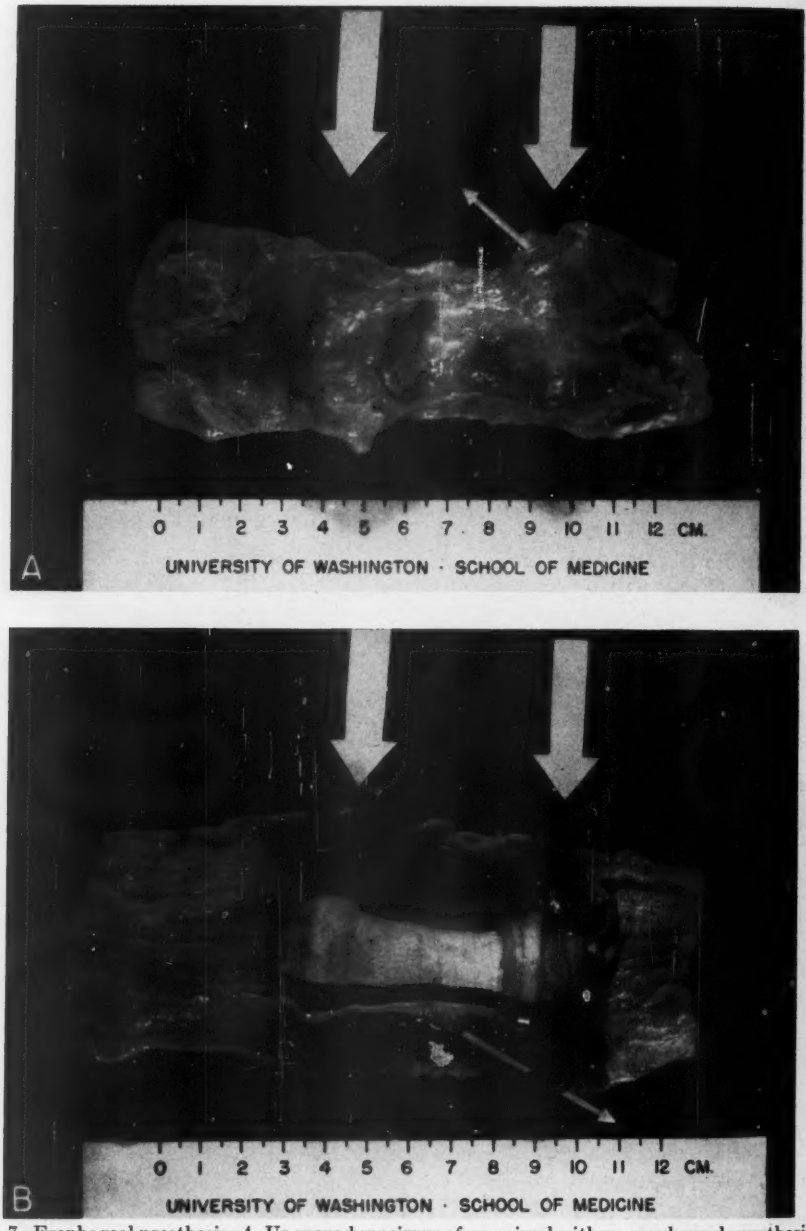
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FIG. 7. Esophageal prosthesis. *A*. Unopened specimen of an animal with an esophageal prosthesis who died of an aortic erosion and rupture of the area indicated by the small arrow (1½ months following operation). *B*. Opened view of the above specimen.

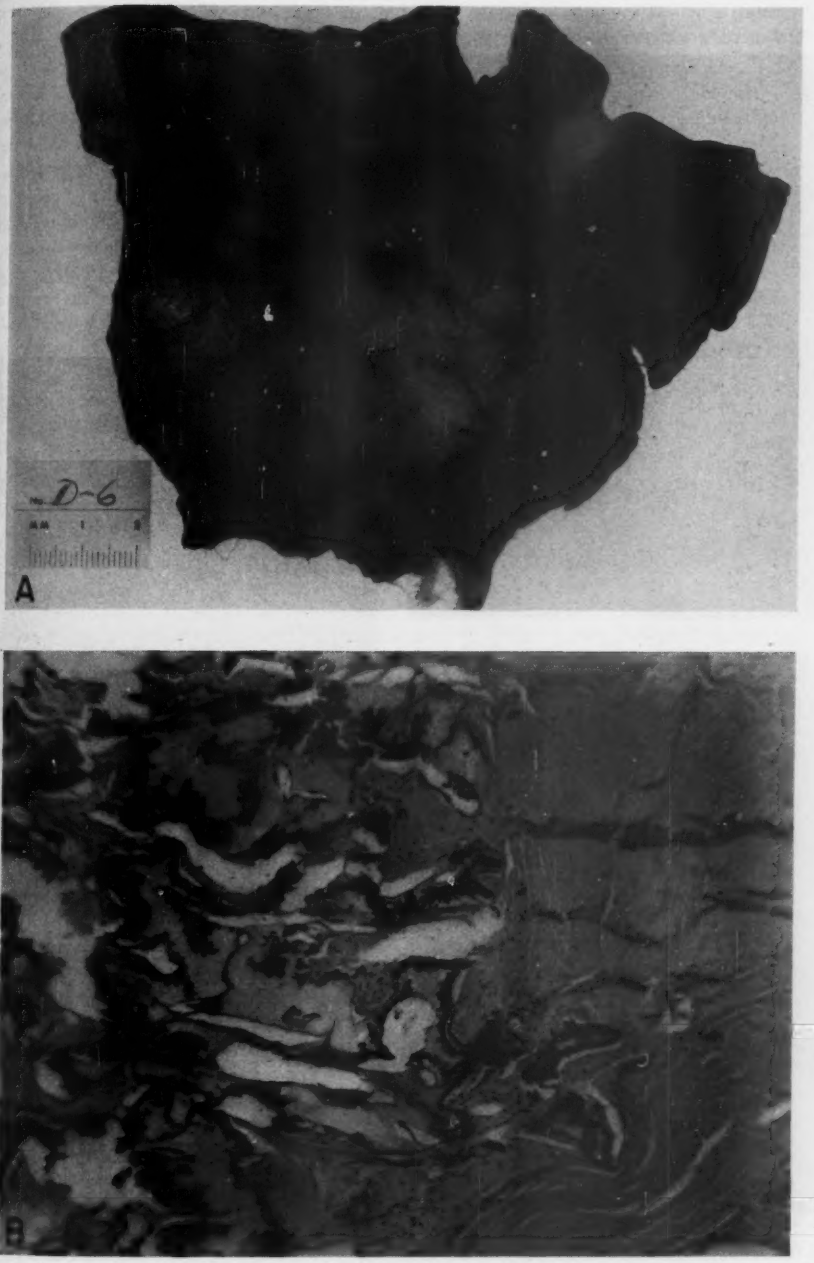

FIG. 8. Ivalon repair of the diaphragm. *A*. Gross specimen. Note the excellent healing without evidence of Ivalon fragmentation or excessive amount of peridiaphragmatic fibrous tissue reaction. *B*. Microscopic section of the above specimen demonstrating the invasion of the Ivalon by fibrous tissue. Note also the excellent healing between the Ivalon and the surrounding edge of the diaphragm.

FIG. 9. Ivalon repair of liver wound. *A*. Illustrating the amount of liver tissue removed (left hemilobectomy). *B*. Ivalon patch repair of the cut liver surface (note hemostasis). *C*. Microscopic section through the area of repair 3 months following operation. Note again the fibrous tissue invasion of the Ivalon from the adjacent liver and the lack of inflammatory or foreign body reaction.

well 8 months after operation. The Ivalon patch when placed over and sutured to the liver and kidney wounds produced an immediate and permanent hemostasis in all cases, with only 1 instance of infection which was an extension of an abdominal wound infection. This animal was alive and well at the end of the 8-month observation period, with complete resolution of the infection. In all cases the Ivalon was invaded by fibrous tissue without evidence of foreign body reaction, cyst formation or other evidence of tissue rejection. None of the wounds were drained and there were no instances of biliary or urinary fistulas (fig. 9).

DISCUSSION

To evaluate its usefulness in surgery, polyvinyl alcohol (Ivalon) sponge has been used in a variety of ways: for small and large arterial replacement, large vein replacement, esophageal and tracheal replacement, a tissue defect replacement and as a hemostatic agent. In general, Ivalon has for all practical purposes little tissue reactivity; however, the experimental work of Oppenheimer should be kept in mind and a word of caution raised because of the fact that malignant tumors can be induced in rodents by the subcutaneous imbedding of Ivalon (Oppenheimer and associates, 1955).[12]

On the other hand, Ivalon is an excellent plastic material to work with technically; it sutures easily and can be molded to any required shape. As emphasized by others (Benjamin and associates, 1957),[2] certain specific precautions should be observed in the construction of aortic grafts of Ivalon. Too much compression of the Ivalon will prevent fibrous tissue invasion of the graft. In the majority of our cases, too little compression was used and this allowed troublesome hemorrhage through the interstices of the sponge and later dilation developed with eventual rupture of the grafts. If these requirements are adhered to, Ivalon can be considered a satisfactory abdominal aortic graft. There is some reservation in our mind as to whether Ivalon should be advocated for thoracic aortic replacement. Certainly compilation grafts of Ivalon are quite satisfactory and in cases in which growth of the graft is a consideration an Ivalon compilation graft could be used. At the present time we are still investigating other materials for vessel replacement, including Orlon, Teflon and chemically modified homografts. Only time will tell whether any of these other materials will be of greater value.

In evaluating the aortic and small artery bypass and pulmonary artery grafts, the only common denominator of failure appeared to be the intraluminal diameter of the graft. There was no correlation as to success or failure of the graft in regard to animal size, graft length or duration after operation. Of the 32 animals in the bypass and pulmonary artery series only 3 received bypass grafts of intraluminal diameters of 7 to 10 mm.; these were all patent at the end of the evaluation period. Twenty-four of the animals had grafts 5 to 8 mm. in diameter; in this group only 3 grafts were patent and 21 were thrombosed. The remaining 5 animals had grafts 4 to 5 mm. in diameter and all of these grafts were thrombosed. In view of the above data and the findings of others we feel that the problem of small artery replacement is still without a satisfactory answer. Ivalon, because of its flexibility and pliability, is of value as a bypass graft when the intraluminal diameter of the graft is 7 mm. or greater. Below 7 mm. in diameter, Ivalon as well as any other material, at the present, is of questionable value as a permanent graft. One should remember, however, the exceedingly important use of compressed Ivalon bypass grafts as temporary shunts in the resection of thoracic aneurysms (DeBakey and associates, 1956).[3] Large vein grafts also present a problem which as yet are not helped by this study. Neither the Ivalon, autogenous vein or autogenous pericardial grafts were of any value; all of the grafts underwent early and complete obstruction by fibrous tissue stricture of the graft. Thus, the problem of blood vessel grafting in areas of low blood flow rates is still unsolved. These factors emphasize the statement that the "smaller the vessel diameter or lower the blood flow rate, the greater the need for excellence in technique of vessel dissection, handling and anastomosis." Edwards and co-workers (1957)[4] have emphasized this point and illustrated that the endothelial lining of the grafts are exceedingly important in the precaution of thrombosis.

The replacement of tracheal segments with Ivalon prosthetics are of limited value. For the patient who requires a palliative tracheal resection for the temporary maintenance of an airway, a properly constructed tracheal prosthetic cer-

tainly will be of some value. The construction of a successful Ivalon esophageal prosthesis is still unsuccessful in our hands.

The use of Ivalon as a hemostatic agent, as an adjunct to the repair of highly vascular organ wounds, is of considerable value and its use in this way can certainly be advocated clinically.

The use of Ivalon patches in the repair of tissue defects is well established, not only by this study with diaphragm repair, but by Pesek and associates (1956).[13] Ivalon patches have also seen considerable use in the repair of intraventricular septal defects (Lewis and Taufic, 1955).[10] The tissue acceptability of Ivalon to tissue defects is well demonstrated by Allen and Webb (1957),[1] who reported on the use of the sponge as successful plombage in patients with active tuberculosis.

SUMMARY

Ivalon can be a valuable aid to the surgeon in the following ways:

1. Abdominal aortic grafts.
2. Compilation aortic grafts in growing patients.
3. Bypass grafts between arteries that are more than 7 mm. in diameter.
4. Bypass grafts as temporary shunts to facilitate aortic surgery.
5. As a hemostatic agent in the repair of liver and kidney wounds.
6. Repair of tissue defects, especially in the diaphragm.

Ivalon can be of limited and temporary value as a tracheal prosthesis.

Ivalon as yet appears to be of no value in the following:

1. Permanent blood vessel grafts less than 7 mm. in diameter.
2. Blood vessel grafts in areas of low blood flow rates (large veins and small arteries).
3. As an esophageal prosthesis.

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PERFORATED DUPLICATION OF ILEUM*

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Duplication of the ileum has been a medical curiosity until recently. Present-day advances in medical care have enabled the doctor to overcome many of the infectious diseases and other common illnesses of children. Developmental anomalies now comprise an important segment of pediatric care.

Duplication of the intestinal tract is an interesting developmental anomaly occasionally found in children of any age. Perforation of such a duplication is an extremely rare complication. Schein and Grundfest[6] have reported recently that a review of the literature reveals only four cases of perforation of an intestinal duplication. In their article they report the fourth such instance. The other three cases were reported by Black,[1] Edwards,[3] and Gross.[4]

We believe that the following case history should be added to the literature as the fifth occasion of this complication, and the third such to recover from the illness.

CASE REPORT

K. M. J. (43798), a white male infant born April 26, 1956, was apparently well until three months of age at which time gastrointestinal bleeding occurred. This was followed one month later by an episode of peritonitis, characterized by fever, prostration, and abdominal distension. On September 9, 1956, a small incision was made in the right lower quadrant, and a large volume of liquid pus (estimated at 1000 to 1500 cc.) was evacuated. The abdomen was not explored at this time because of the gross infection present. Penrose drains were placed through the incision, and the wound was closed around them. After a brief period of purulent drainage, a fecal fistula developed which closed spontaneously approximately one month later.

The patient was discharged home on October 21 for one month, and appeared in good condition on his return. Shortly after admission, however, an abscess developed in the healed incision and was drained, with good response. At about this same time, the patient passed black stools for three days which tested positive for blood. His hemoglobin decreased from 14.1 gm. per cent to 6.4 gm. per cent.

Laparotomy was performed on December 10, at which time the patient was eight months old. The abdomen was entered through a left transverse incision to avoid the right lower quadrant wound and sinus. Multiple adhesions held loops of small intestine together to form a 2-cm. diameter abscess near the original incision and connected to the old wound by an open sinus tract. The appendix was not a part of this abscess, and appeared normal. The loops of bowel were separated carefully, and a duplication of the ileum was found. The base of the duplication had healed but formed an integral part of the abscess wall. The base of the duplication was approximately 25 cm. proximal to the ileocecal valve, and opened into the bowel lumen. It measured 30 cm. long and for two thirds of this length shared muscular wall in common with the apparently normal ileum, then extended freely within the mesentery. The duplication and adjacent ileum were resected and an end-to-end anastomosis done. The patient recovered promptly and within a few days was eating well.

The pathology report (Dr. Israel Diamond, Louisville Children's Hospital) described the specimen grossly as above. The lining of the duplication was gastric type mucosa throughout.

Eight months after discharge the patient suffered a severe gastrointestinal hemorrhage necessitating transfusion; this was done at his local hospital. Two months later a second episode occurred which also required transfusion and following this the family consented to return the patient to the Children's Hospital for exploration of a shadow in the left thorax which had been noted at the time of his initial hospitalization. Ten and a half months after discharge from the hospital a thoracotomy was performed and a duplication of the stomach was removed from the left thorax. This duplication had no direct communication with any part of the gastrointestinal tract, but gastric mucosa and pyloric canal were easily identified. The youngster made a rapid recovery and was discharged from the hospital 16 days later. Since October 1957 there has been no recurrence of intestinal bleeding.

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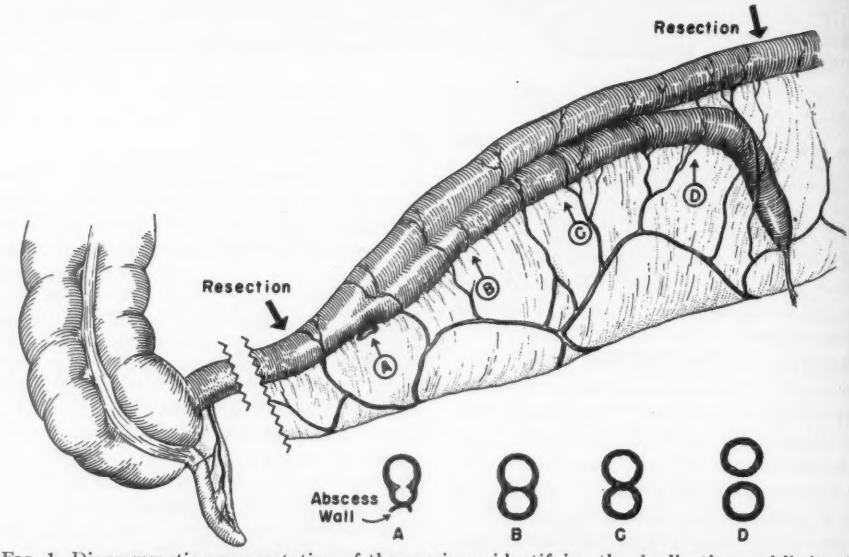

FIG. 1. Diagrammatic representation of the specimen identifying the duplication and limits of resection. Below are cross-section diagrams to illustrate the configuration of the specimen.

FIG. 2. Photograph of opened specimen showing resected ileum with its duplication attached below.

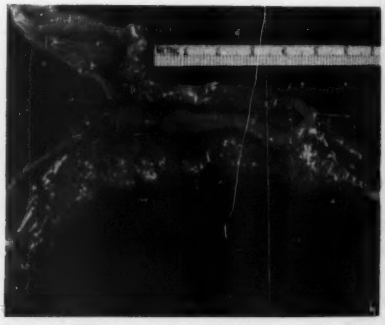

FIG. 3. Photograph showing connection of duplication to ileum as identified by probe within opened duplication.

DISCUSSION

Two complications of intestinal duplication were present in this infant, hemorrhage and perforation. Certainly the patient's strong constitution was necessary for his recovery.

Gross[5] describes these complications as well as obstruction and pain. In a series of 68 alimentary duplications, he reports 19 associated with the ileum, one of which was perforated.

Duplication may occur in any region of the alimentary tract, but is most common in the ileum. The lining mucosa need not be typical of that region but may resemble any other epithelium of the alimentary tract.

The etiology for such formations is not understood, but the mechanism is thought to be along the lines of Bremer's theory.[2] In the embryo, the alimentary tract is represented by the fore-, mid-, and hind-gut, communicating with the yolk sac. As development progresses, epithelial proliferation converts the tract to a solid cord of cells. Later, the lumen appears spontaneously in different places which then become connected as a patent lumen. It is likely that improper align-

ment of this final lumed is the manner of development of this condition

Aberrant gastric mucosa has been reported in esophagus and Meckel's diverticulum as well as other regions of the alimentary tract. Peptic ulcer sometimes results near this acid producing mucosa. This is probably the reason for perforation and/or bleeding in duplication of the ileum. Undoubtedly, many duplications occur which are never discovered because they cause no symptoms.

The treatment of bowel duplications is surgical excision of the duplication and associated normal bowel, since the blood supply is usually common to both.

SUMMARY

A case of duplication of the ileum complicated by perforation and hemorrhage is presented, with a brief discussion of the condition.

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EDITORIAL

DEATH OF AN INSTITUTION

The first blood vessel banks were established almost 10 years ago for the purpose of providing a supply of arterial homografts for clinical use. In the beginning homografts were employed primarily in the treatment of coarctation of the aorta and acute arterial injuries. Later, development of operations for aortic aneurysms and for atherosclerotic occlusion of the aorta and peripheral arteries extended the indications for vascular replacement, and blood vessel banks became commonplace even in community hospitals.

Arterial homografts were preserved in a variety of ways. Early preservation was accomplished by quick freezing or by refrigeration in a nutrient solution. With both of these methods, however, it was necessary to remove the homografts under aseptic conditions and even then the danger of contamination was great. In addition, the length of time the homografts could be preserved was limited. For these reasons, other techniques for preservation were developed. In a few institutions where supervoltage x-ray equipment was available, grafts were sterilized by irradiation prior to freezing. The method most widely employed consisted in sterilization of fresh homografts by immersion in ethylene oxide followed by lyophilization. Grafts processed in this manner could be kept at room temperature for an indefinite period of time. Later, sterilization by treatment with beta propiolactone was introduced, thus increasing the safety of preservation by refrigeration.

In 1954 the clinical use of preserved arterial homografts was well established and blood vessel banks were operating effectively. Nevertheless, one of the chief disadvantages of the natural arterial replacement still remained; namely, that in order for a homograft to become available someone had to die. In metropolitan centers where accidental and homicidal deaths were frequent, procurement was no problem; but in the smaller, more law abiding communities it was a serious problem indeed.

Concurrent with the development of methods for preserving arterial homografts, there was investigation of synthetic materials for blood vessel replacement. Thus, by the time blood vessel banks were well established, synthetic substitutes had been studied extensively in the experimental animal and had been employed in a few clinical cases. Early in 1954 Edwards and Tapp introduced an arterial graft made of braided nylon. The nylon tube was treated with formic acid to reduce its porosity and was then crimped to produce flexibility. These features made the Edwards-Tapp prosthesis more satisfactory for arterial replacement or bypass, particularly in the lower extremities, than any other prosthesis previously described. Subsequently, these and other types of synthetic vascular substitutes have become available commercially.

Sufficient experience has accumulated with synthetic prostheses to permit their comparison with arterial homografts. The prostheses are readily available, can be sterilized by autoclaving, and, if utilized under proper circumstances, they function as satisfactorily as homografts. They are resistant to infection and even when exposed will continue to function for a long time. Their chief disadvantage is porosity, which requires "preclotting" to reduce bleeding through the walls.

It is apparent, therefore, that blood vessel replacements made of synthetic material are as satisfactory functionally as arterial homografts and have certain advantages which homografts do not possess. Because of these advantages plastic prostheses are rapidly replacing arterial homografts in the treatment of vascular lesions. And with the homograft will go the blood vessel bank—a short lived institution, to be sure, but one which has played a vital role in the development of vascular surgery in the past decade.

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BOOK REVIEWS

The editors of THE AMERICAN SURGEON *will at all times welcome new books in the field of surgery and will acknowledge their receipt in these pages. The editors do not, however, agree to review all books that have been submitted without solicitation.*

Plastic Arterial Graft. By W. STERLING EDWARD, Assistant Professor of Surgery, Medical College of Alabama. Charles C Thomas, Publisher, Springfield, Ill., 1957.

The field of vascular surgery, while well established, is relatively one of the younger branches of surgery. Increasing recognition of arterial vascular problems, better diagnostic modalities and, finally, knowledge of the organ and tissue tolerance under conditions of interrupted arterial flow have made direct surgical attack on the major vascular system a relatively safe and common occurrence. Success in vascular surgery would have been impossible without fundamental research on and constant search for a suitable vascular substitute.

This volume, *Plastic Arterial Grafts*, must of necessity be in the nature of a progress report. It is a timely and informative volume. The book is divided into six chapters and each chapter is followed by an extensive bibliography. The section on the history of arterial grafts is thoroughly enjoyable. It is followed by an interesting account of cooperative effort by the industrial and medical disciplines, and the constant efforts, trials and errors of the author and his group before chemically treated crimped nylon was developed. In subsequent chapters, problems of arterial injury, segmental arterial occlusion, aneurysm and the scheme of operation are discussed. The last chapter is devoted to unsolved problems in the field of arterial replacement. The volume should prove of interest to all general surgeons.

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Operative Surgery, Vol. 4. Edited by CHARLES ROB, M.C., M.CHIR., F.R.C.S., AND RODNEY SMITH, M.S., F.R.C.S. Butterworth & Co., Ltd., London, 1957.

Volume 4 of *Operative Surgery*, under the general editorship of Charles Rob and Rodney Smith, is written by outstanding British authorities. This volume consists of three parts. Part one comprises operations on the head (excluding cranial procedures), neck, face, jaw, as well as axillary and inguinal lymph node dissection. Part two is devoted to vascular surgery, including thoracic and lumbar sympathetic ganglionectomy. An adequate description of such procedures as aortography, arteriography and venography is included. The final part covers operative procedures on the endocrine glands, *e.g.*, thyroid, parathyroid, thymus, pituitary and adrenal glands. The procedures described are similar to those employed in this country. The book is well illustrated and there is adequate explanatory material accompanying each illustration. Each section is followed by a brief and up-to-date bibliographic note. The format and printing are excellent.

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